EVALUATION OF RESPIRATORY IMPAIRMENT BY PULMONARY FUNCTION TEST IN AUTODRIVERS

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

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for the award of the degree of

M.D. (PHYSIOLOGY) BRANCH – V

CHENGALPATTU MEDICAL COLLEGE,

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI – TAMILNADU

OCTOBER 2018

CERTIFICATE

This is to certify that this dissertation titled **"EVALUATION OF RESPIRATORY IMPAIRMENT BY PULMONARY FUNCTION TEST IN AUTODRIVERS''** is a bonafide record work done by **Dr. P.AMUTHAMOZHI,** during the period of her postgraduate study from 2015 to 2018 under guidance and supervision in the Department of Physiology, Chengalpattu Medical College and Hospital, Chengalpattu – 603 001 in partial fulfillment of the requirement for **M.D. PHYSIOLOGY** degree Examination of The Tamil Nadu Dr. M.G.R. Medical University.

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DECLARATION

I declare that the dissertation entitled **"EVALUATION OF RESPIRATORY IMPAIRMENT BY PULMONARY FUNCTION TEST IN AUTO DRIVERS"** submitted by me for the degree of M.D. is the record work carried out by me during the period of **March 2017 to October 2017** under the guidance of **Dr. A. ANITHA, M.D., DCH.,** Head of the Department of Physiology, Chengalpattu Medical College, Chengalpattu. This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the University regulations for the award of degree of M.D., Physiology (Branch V) examinations to be held in October 2018.

Place: Chengalpattu **Signature of the Candidate**

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LIST OF ABBREVIATIONS

WHO	-	World Health Organization
PM10	-	Particulate matter of 10 microns or less in diameter
UNEP	-	United Nation Environment Programme
USA	-	United States of America
CNG	-	Compressed Natural Gas
LPG	-	Liquefied Petroleum Gas
NAMP	-	National Air Quality Monitoring Programme
SO_2	-	Sulphur-di-oxide
NO_2	-	Nitrogen-di-oxide
RSPM	-	Respirable Suspended Particulate Matter
SPM	-	Suspended Particulate Matter
COPD	-	Chronic Obstructive Pulmonary Disease
CVD	-	Cardiovascular Disease
DEP	-	Diesel Exhaust Particles
PMN	-	Polymorph Nuclear Cells
IUGR	-	Intra Uterine Growth Retardation
NAAQS	-	National Ambient Air Quality Standards

INSTITUTIONAL ETHICAL COMMITTEE

CHENGALPATTU MEDICAL COLLEGE, CHENGALPATTU

Title of Work	: Evaluation of respiratory impairment by pulmonary function Test in Auto Drivers.
Principal Investigator	: Dr.Amuthamozhi
Designation	: 1st Year Physiology
Co-Investigators	: Dr.Anitha,MD,.DCH,. Professor of Physiology Dr.G.Ravikumar, MD,.(TD&RD) Asst. Professor of Thoracic Medicine
Department	: Physiology

The request for an approval From the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 11.05.2016 at the Medical Education Unit, Government Chengalpattu Medical College, Chengalpattu at 12.00 PM.

The Members of the committee, the Secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal Investigator and their team are directed to adhere to the guidelines given below:

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4. You should abide to the rules and regulations of the institution(s).
5. You should complete the work within the specific period and if any extension is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on complete of work.

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IEC, CHENGALPATTU MEDICAL COLLEGE
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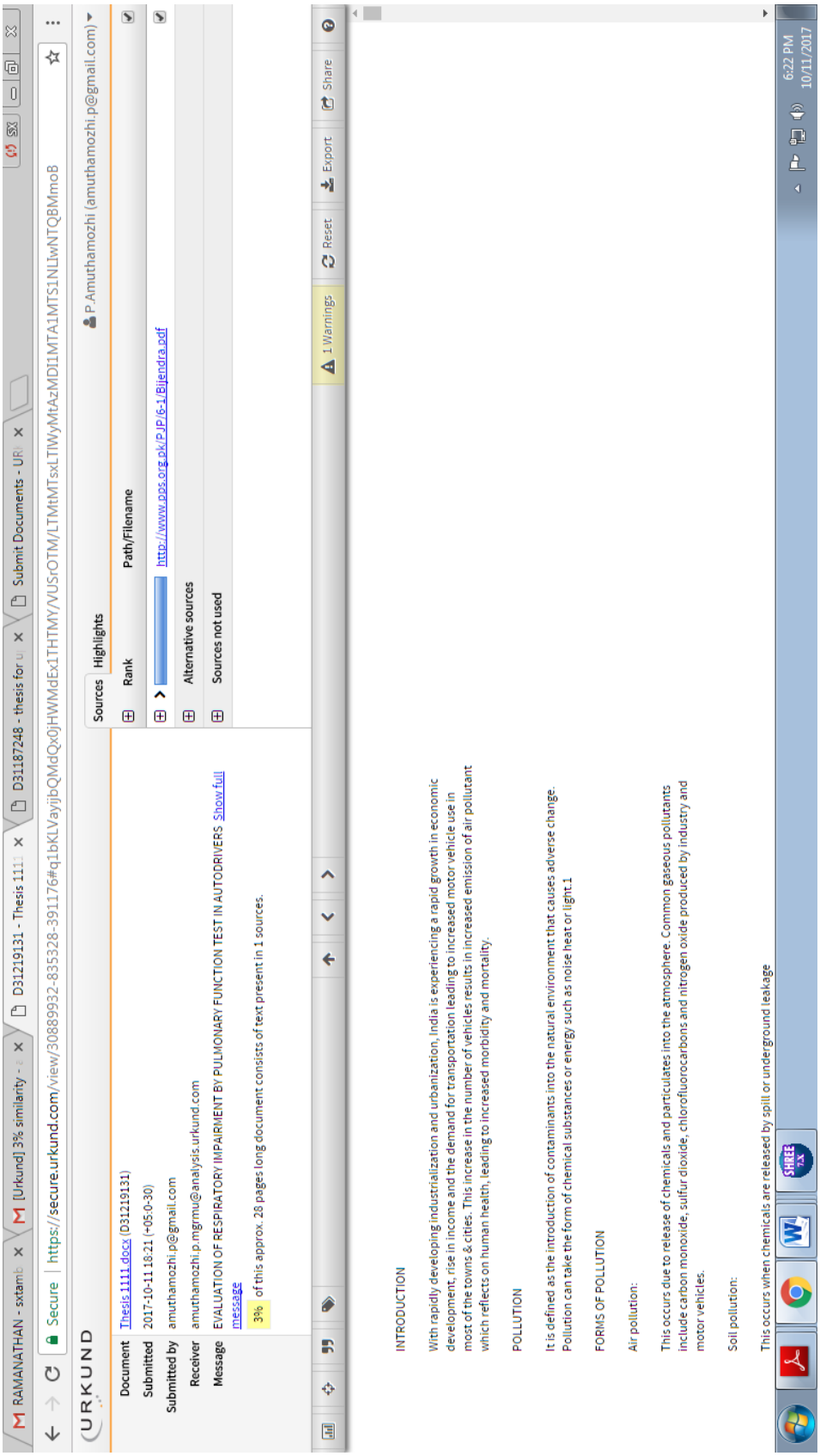
PLAGIARISM CERTIFICATE



CERTIFICATE - II

This is to certify that this dissertation work titled "**EVALUATION OF RESPIRATORY IMPAIRMENT BY PULMONARY FUNCTION TEST IN AUTO DRIVERS**'' of the candidate **DR.P.AMUTHAMOZHI** with registration Number **201515501** for the award of degree of **M.D.** in the branch of **PHYSIOLOGY - BRANCH – V.** I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 3**%** percentage of plagiarism in the dissertation.

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INTRODUCTION

With rapidly developing industrialization and urbanization, India is experiencing a rapid growth in economic development, rise in income and the demand for transportation leading to increased motor vehicle use in most of the towns & cities. This increase in the number of vehicles results in increased emission of air pollutant which reflects on human health, leading to increased morbidity and mortality.

POLLUTION

It is defined as the introduction of contaminants into the natural environment that causes adverse change. Pollution can take the form of chemical substances or energy such as noise heat or light.[1]

FORMS OF POLLUTION

Air pollution:

This occurs due to release of chemicals and particulates into the atmosphere. Common gaseous pollutants include carbon monoxide, sulfur dioxide, chlorofluorocarbons and nitrogen oxide produced by industry and motor vehicles.

Soil pollution:

This occurs when chemicals are released by spill or underground leakage

in to the soil. The most significant soil contaminants are hydrocarbons, heavy metals, herbicides, pesticides and chlorinated compounds.

Water pollution:

This occurs by the discharge of waste water from commercial and industrial waste into surface waters, such as chlorine from untreated sewage and agricultural runoff, which may contain chemical fertilizers and pesticides.

Light pollution:

This includes light trespass, over-illumination etc.

Noise pollution:

This includes roadway noise, aircraft noise, industrial noise as well as high-intensity sonar.

Thermal pollution:

This occurs due to temperature change in natural water bodies caused by human influence, such as use of water as coolant in a power plant.

Plastic pollution:

This includes the accumulation of plastic products in the environment that adversely affects wildlife, wildlife habitat, or humans.

Radioactive contamination:

This results from 20th century activities in atomic physics, such as nuclear power generation and nuclear weapons research, manufacture and deployment.[2]

AIR POLLUTION

There are various types of pollution of which air pollution is our major concern. Air pollution is defined as a mixture of natural and man-made substances in the air we breathe. It is typically separated into two categories:

1. Outdoor air pollution
2. Indoor air pollution.

1. Outdoor air pollution:

It involves exposures that take place outside of the built environment. Examples include:

- Fine particles produced by the burning of fossil fuels (i.e. the coal and petroleum used in traffic and energy production)
- Noxious gases (sulfur dioxide, nitrogen oxides, carbon monoxide, chemical vapors, etc.)
- Ground-level ozone (a reactive form of oxygen and a primary component of urban smog)
- Tobacco smoke

2. Indoor air pollution:

It involves exposures to particulates, carbon monoxides, and other pollutants carried by indoor air or dust. Examples include:

- Gases (carbon monoxide, radon, etc.)
- Household products and chemicals
- Building materials (asbestos, formaldehyde, lead etc.)
- Outdoor indoor allergens (cockroach and mouse dropping, etc.)
- Tobacco smoke
- Mold and pollen[3]

WORLD HEALTH ORGANIZATION (WHO) GUIDELINES

"WHO Air Quality Guidelines" estimate that reducing annual average particulate matter (PM_{10}) concentrations from levels of 70μg/m^3, common in many developing cities, to the WHO guideline level of 20μg/m^3, could reduce air pollution-related deaths by around 15%. There are serious risks to health not only from exposure to PM, but also from exposure to ozone (O_3), nitrogen dioxide (NO_2) and sulfur dioxide (SO_2).

WHO estimates that in 2012, some 72% of outdoor air pollution-related premature deaths were due to ischemic heart disease and strokes, while 14% of deaths were due to chronic obstructive pulmonary disease or acute lower respiratory infections and 14% of deaths were due to lung cancer. This mortality

is due to exposure to small particulate matter of 10 microns or less in diameter (PM_{10}), which causes cardiovascular and respiratory disease, and cancers.[4]

Outdoor air pollution is a major environmental health problem affecting everyone in developed and in developing countries. The WHO has estimated that urban air pollution is responsible for approximately 800,000 deaths and 4.6 million lost life-years each year around the globe (WHO, 2002). The constituents of air pollution in different parts of the world are largely similar, but the magnitude of exposure, general health status of the people, nutritional and other disparities and the level of health care facilities are different across the globe.[5]

HISTORY OF AIR POLLUTION

Air pollution is recognized as a major threat to human health. The United Nations Environment Programme has estimated that globally 1.1 billion people breathe unhealthy air (UNEP, 2002). Epidemiological studies have shown that concentrations of ambient air particles are associated with a wide range of effects on human health, especially on the cardio-respiratory system.[6]

Our concern about air pollution and its effect on human health is concerned primarily from three major episodes of air pollution - Meuse Valley of Belgium in 1930, Donora in Pennsylvania of USA in 1948, and London smog episode in 1952. These episodes prompted many countries in Europe and North America to initiate regulatory measures to control outdoor air pollution.

The London fog incident in 1952 conclusively established an association between air pollution and increased mortality.[7] Since then, several epidemiological studies were done in a short period of six years from 1989 to 1995, which unrevealed the role of particulate matter as the chief mediator of toxic effects of air pollution. These studies in the USA and Europe have established a clear relationship between air pollution exposure and excess mortality especially due to cardio respiratory diseases.[8 & 9.]

URBANIZATION AND AIR POLLUTION IN INDIA

Air pollution in Asian cities is closely related to levels and trends in economic and social development. India is experiencing a rapid growth with population size and economic activity, reflected by industrialization and urbanization in recent times. Currently about two-third of Indian population is living in rural areas. But the pattern is changing rapidly as more people are moving towards the cities in search of livelihood.

The Metropolitan area comprises of the city and its outlying urban and rural areas. With good infrastructure facilities, the urban cities have become a major center for commerce, industry and education. Our study area belongs to semi urban area with rapid urbanization and need for transportation there is increase in number of vehicles running on fuels. This fuel on combustion contributes to air pollution.

SOURCES OF URBAN AIR POLLUTION

Air pollution is identified as a matter of environmental concern in all metropolitan cities. With the increasing commercial and industrial activities, the transport system is also increasing day by day in urban cities leading to deterioration in air quality. In cities air pollution is contributed by the following sectors

1. Vehicular sources
2. Industrial sources
3. Domestic sources
4. Other sources

VEHICULAR SOURCES

In general, combustion is the chief contributor to outdoor air pollution. In most cities, the major source of combustion is fuel use. In the last three decades, the number of motorized vehicles in India has increased 29-times, from 1.9 million in 1971 to 55.0 million in 2001 .The increase was not uniform for all vehicle types: it was 7-fold for buses, 9-fold for trucks, 10-fold for car, jeeps and taxis, but a remarkable 67-fold for two-wheelers.[10]

Motor vehicles have internal combustion engine which burns a mixture of air and fuel. The type and quantity of the pollutants released during this combustion is influenced by more than a dozen factors. The kind of fuel- petrol, diesel or compressed natural gas (CNG) is just one of them. However, fuel type

is a useful indicator of potential emissions. Coal and biomass are high emitting solid fuels, petrol diesel and kerosene are mid-emitting liquid fuels and liquefied petroleum gas (LPG) and CNG are low-emitting gaseous fuels.

Transport sector consumes half of the petroleum products in the world, and same is true for India. Currently vehicular pollution contributes to 72% of total air pollution in major cities.[11]

Ambient Air Quality

Ambient air quality is carried out in urban and semi urban cities by Tamil Nadu State Pollution Control Board and National Environmental Engineering Research Institute. The monitoring is carried out under National Air Quality Monitoring Programme (NAMP).

Air quality with respect to SO_2, NO_2, SPM and RSPM has been determined in terms of low, moderate, high and critical levels. In general high levels of SO_2 and NO_2were observed in residential areas and industrial areas of urban and semi urban areas cities.[12, 13 & 14.]

Air Quality Trends in urban cities

Trend in annual average concentration of SO2 in residential areas and industrial areas is depicted in Figure no: 1. The SO2 levels were higher than the NAAQS (annual average) during all the monitored years in industrial areas than in residential areas. Trend in annual average concentration of NO2 in residential areas and industrial areas is depicted in Figure no: 2. NO2 levels were lower than

the NAAQS (annual average) in residential areas than industrial areas during all the monitored years. Trend in annual average concentration of RSPM in residential areas and industrial areas is depicted in Figure no: 3. RSPM levels were lower than the NAAQS (annual average) during many years in residential areas than industrial areas. Trend in annual average concentration of SPM in residential areas and industrial areas is depicted in Figure no: 4. SPM levels were lower than the NAAQS (annual average) during many years in residential areas and industrial area

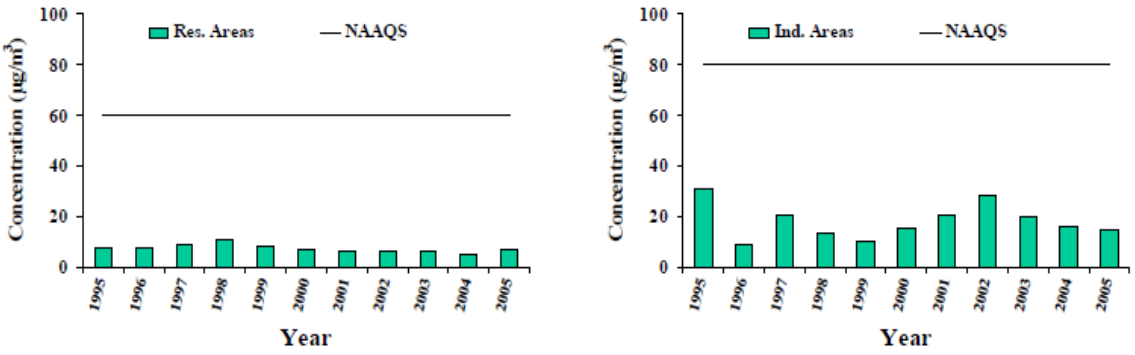

Figure no: 1 Trend in annual average concentration of SO2 in residential areas and industrial areas.

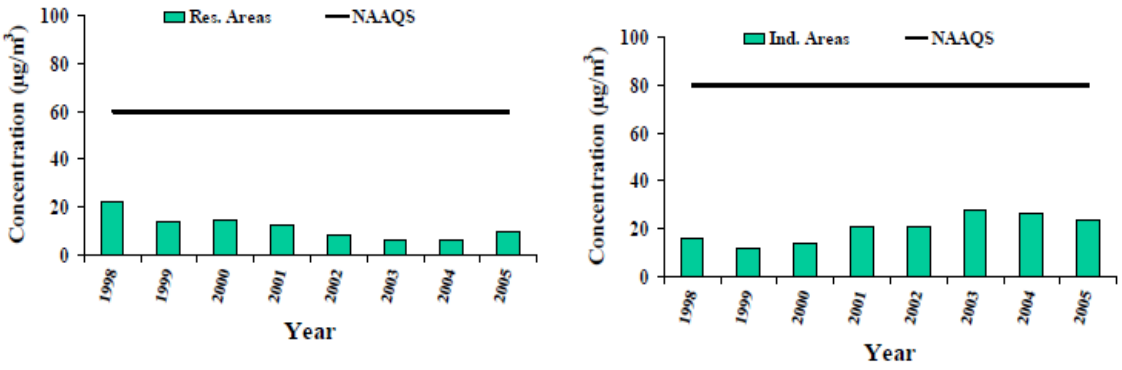

Figure no: 2 Trend in annual average concentration of NO2 in residential areas and industrial areas.

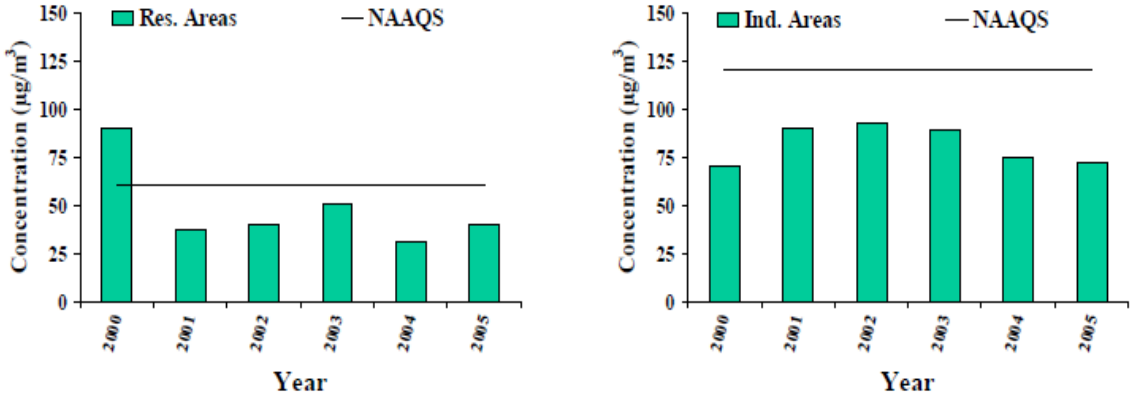

Figure no: 3 Trend in annual average concentration of RSPM in residential areas and industrial areas.

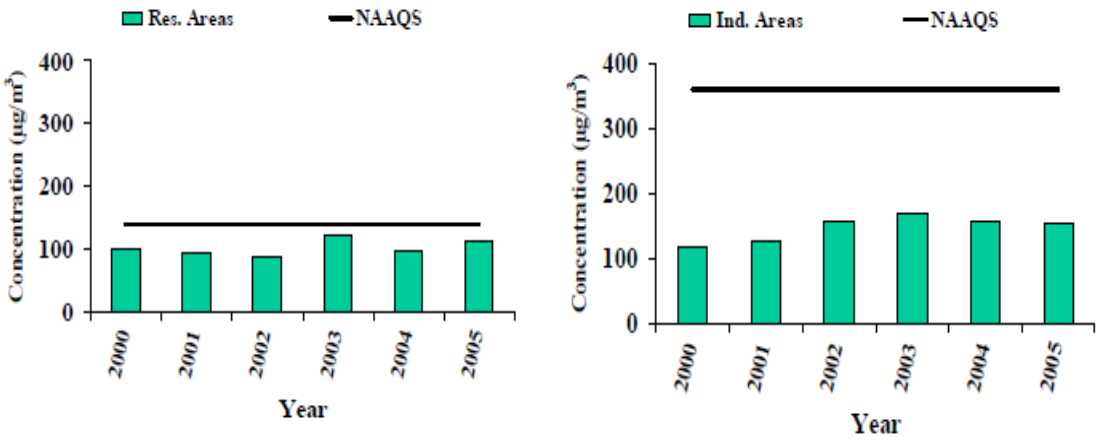

Figure no: 4 Trend in annual average concentration of SPM in residential areas and industrial areas.[15,16 &17.]

HEALTH EFFECTS OF AIR POLLUTION

Harmful effects of air pollution on human health are recognized for centuries. It has been estimated that globally 8,000 people die every day from diseases related to air pollution exposure. Each year 60,000 deaths in the United States and 500,000 deaths in China occur due to air pollution. Several epidemiological studies have established a direct relationship between the pollutants and health hazards ranging from morbidity (illness) to mortality (death from illness).

SYSTEMIC EFFECTS OF AIR POLLUTION

Mortality and morbidity associated with air pollution are primarily due to the toxic effects of particulates.

(a) Cardiovascular changes

Reports indicate that air pollution may affect blood pressure. High blood pressure (hypertension) is common among persons exposed to high level of air pollution.[18] Elevated plasma viscosity, increased heart rate (>80 beats/min), reduced heart rate variability and increased risk of arterial hypertension have been reported in association with chronic air pollution exposure.[19]

Air pollution exposure and cardiovascular diseases (CVD) are intimately related, and it is a growing concern worldwide. CVD associated with air pollution is angina, cardiac insufficiency, hypertension and myocardial infarction. Studies conducted from the late nineties have consistently shown that PM10 is associated with overall hospital admissions for CVD.[20]

Air pollution has been associated with sudden death in patients with stable angina and myocardial infarction. Among air pollution-related deaths in the US in 1997, only 8.5% were from respiratory diseases (COPD, pneumonia, influenza etc.) while cardiovascular deaths (heart, cerebrovascular and arterial diseases) accounted for 39.5% of all deaths.[21]Thus, CVD, along with respiratory ailments, are the most important cause of death from air pollution exposure.

b) Hematotoxicity of air pollution

DEP is a potent stimulus for release of neutrophils from the bone marrow and the transit from blood to the airway tissues. Acute exposure to ambient particles accelerates the transit of PMN from marrow to the circulation, whereas chronic exposure expands the size of the bone marrow pool of PMN. Volatile organic compounds (benzene, toluene and xylene) are haematotoxic, and exposures to these pollutants are associated with higher prevalence of hematological abnormalities like alterations in WBC, RBC and platelet count in children.[22]

Benzene cause bone marrow suppression, decreased erythrocyte, hemoglobin and hematocrit levels leading to anemia, suppression of WBC counts (leukopenia), and reduction in platelet number (thrombocytopenia).

(b) Immunotoxicity of air pollution

Following DEP exposure among human volunteers, T-lymphocytes, mostly CD4+ cells, infiltrate the submucosa and bronchial epithelium. Chronic exposure to vehicular pollution is associated with airway inflammation, and diesel exhaust particles play an important role in this response. Oxidative stress induced by these free radicals increases the permeability of epithelial cells that further facilitate the transfer of particles into the Interstitium.[23]

Reproductive toxicity

Particulate matter can significantly increase the adverse reproductive outcomes in both males and females. Studies show relatively high level of air pollution result in intrauterine growth retardation (IUGR) in the first gestational month in females and YY8 disomic in the sperms.[24]

(c) Neurotoxicity

Besides physical health, air pollution exposure may lead to impairment of mental health, because toxic effects of particulate matters on central and peripheral nervous system has been reported. Difficulties with recall, response, concentration, and sleep disorders suggest central nervous system impairment due to vehicular emission.[25]

(d) Genotoxic effects of air pollution

Exposure to vehicular emission may cause genetic changes as long-term adverse health effect. Urban atmospheres contain complex mixtures of air pollutants including mutagenic and carcinogenic substances such as benzene, diesel soot and heavy metals etc.[26] Different chemical agents or their metabolites may cause DNA strand breaks, impairment of DNA repair system, dysregulation of cell cycle.

EFFECT OF AIR POLLUTION ON RESPIRATORY SYSTEM

RESPIRATORY SYSTEM

The organ that supports gas exchange comprises the respiratory system. They are the upper airways, lower airways, lung parenchyma, chest wall, respiratory muscle, pulmonary blood vessels, support nerves and lymphatics.

Lungs are multilobed, cone shaped, sponge like organs that lie within the pleural cavities bounded by chest wall & diaphragm. The average adult lungs are low – density organs that occupy a volume of approximately 3.5 liters and weight approximately 900gm.

Respiration, as the term is generally used, includes two processes: **External respiration,** the absorption of O_2 and removal of CO_2 from the body as a whole; and **Internal respiration,** the utilization of O_2 and production of CO_2 by cells and the gaseous exchanges between the cells and their fluid medium.

ANATOMY OF THE LUNGS

The respiratory system is made up of a gas-exchanging organ (the lungs) and a "pump" that ventilates the lungs. The pump consists of the chest wall; the respiratory muscles, which increase and decrease the size of the thoracic cavity; the areas in the brain that control the muscles; and the tracts and nerves that connect the brain to the muscles.

At rest, a normal human breathes 12 to 15 times a minute. About 500 mL of air per breath, or 6 to 8 L/min, is inspired and expired. This air mixes with the

gas in the alveoli, and, by simple diffusion, O_2 enters the blood in the pulmonary capillaries while CO_2 enters the alveoli. In this manner, 250 mL of O_2 enters the body per minute and 200 mL of CO_2 is excreted.

Air Passages

After passing through the nasal passages and pharynx, where it is warmed and takes up water vapor, the inspired air passes down the trachea and through the bronchioles, respiratory bronchioles, and alveolar ducts to the alveoli, where gas exchange occurs. Between the trachea and the alveolar sacs, the airways divide 23 times.

The first 16 generations of passages form the conducting zone of the airways that transports gas from and to the exterior. They are made up of bronchi, bronchioles, and terminal bronchioles. The remaining seven generations form the transitional and respiratory zones where gas exchange occurs; they are made up of respiratory bronchioles, alveolar ducts and alveoli.

These multiple divisions greatly increase the total cross-sectional area of the airways, from 2.5 cm^2 in the trachea to 11,800 cm^2 in the alveoli. Consequently, the velocity of air flow in the small airways declines to very low values.

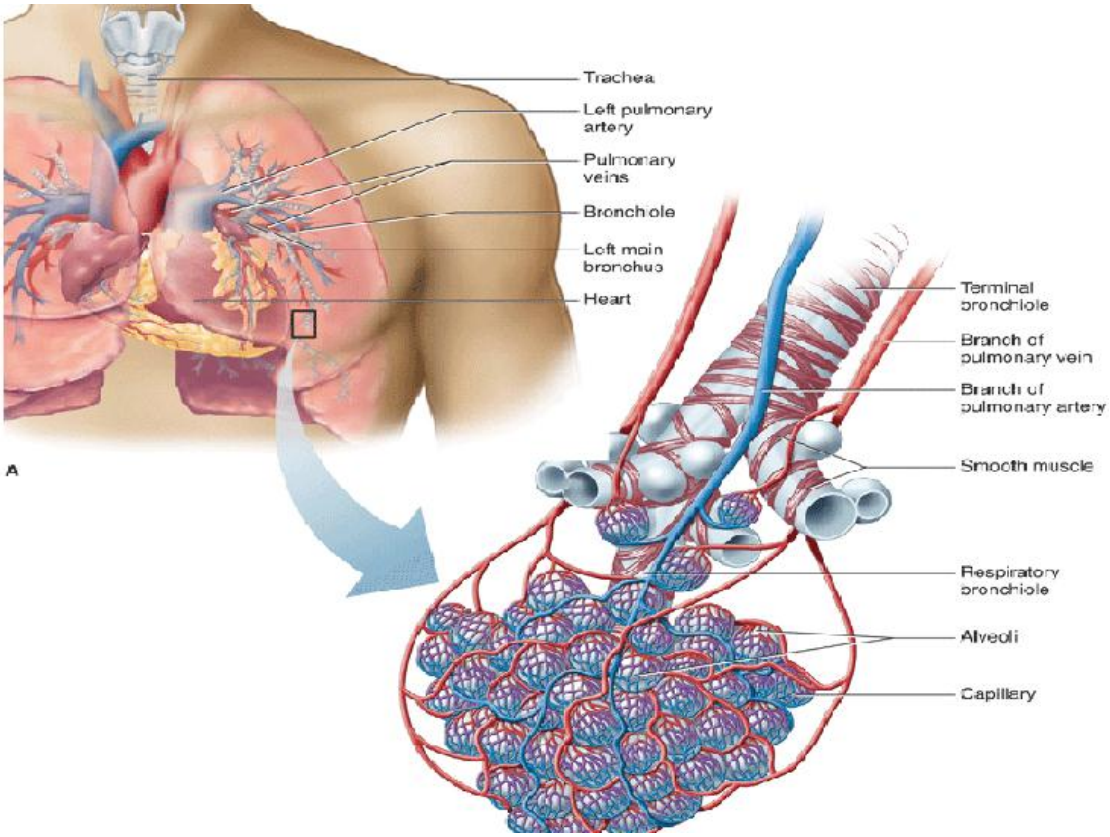

Figure no.5 lung and respiratory passage

Inspiration & Expiration

The lungs and the chest wall are elastic structures. Normally, no more than a thin layer of fluid is present between the lungs and the chest wall (intrapleural space). The lungs slide easily on the chest wall, but resist being pulled away from it in the same way that two moist pieces of glass slide on each other but resist separation.

The pressure in the "space" between the lungs and chest wall (intrapleural pressure) is sub-atmospheric. The lungs are stretched when they expand at birth and at the end of quiet expiration their tendency to recoil from the chest wall is just balanced by the tendency of the chest wall to recoil in the opposite direction.

If the chest wall is opened, the lungs collapse; and if the lungs lose their elasticity, the chest expands and becomes barrel-shaped.

Inspiration is an active process. The contraction of the inspiratory muscles increases intrathoracic volume. The intrapleural pressure at the base of the lungs, which is normally about –2.5 mm Hg (relative to atmospheric) at the start of inspiration, decreases to about –6 mm Hg.

The lungs are pulled into a more expanded position. The pressure inside the airway becomes slightly negative, and therefore air flows into the lungs. At the end of inspiration, the lung recoil begins to pull the chest back to the expiratory position, where the recoil pressures of the lungs and chest wall balance.

The pressure in the airway becomes slightly positive, and air flows out of the lungs. Expiration during quiet breathing is passive. However, some contraction of the inspiratory muscles occurs in the early part of expiration. This contraction exerts a braking action on the recoil forces and slows expiration.

Lung Volumes

- The amount of air that moves into the lungs with each inspiration (or the amount that moves out with each expiration) is called the **tidal volume.**
- The air inspired with a maximal inspiratory effort in excess of the tidal volume is the **inspiratory reserve volume.**
- The volume expelled by an active expiratory effort after passive expiration is the **expiratory reserve volume.**
- The air left in the lungs after a maximal expiratory effort is the **residual volume.**
- The space in the conducting zone of the airways occupied by gas that does not exchange with blood in the pulmonary vessels is the **respiratory dead space.**
- The amount of air inspired per minute **(pulmonary ventilation, respiratory minute volume)** is normally about 6 L (500 mL/ breath x 12 breaths/min).[27]

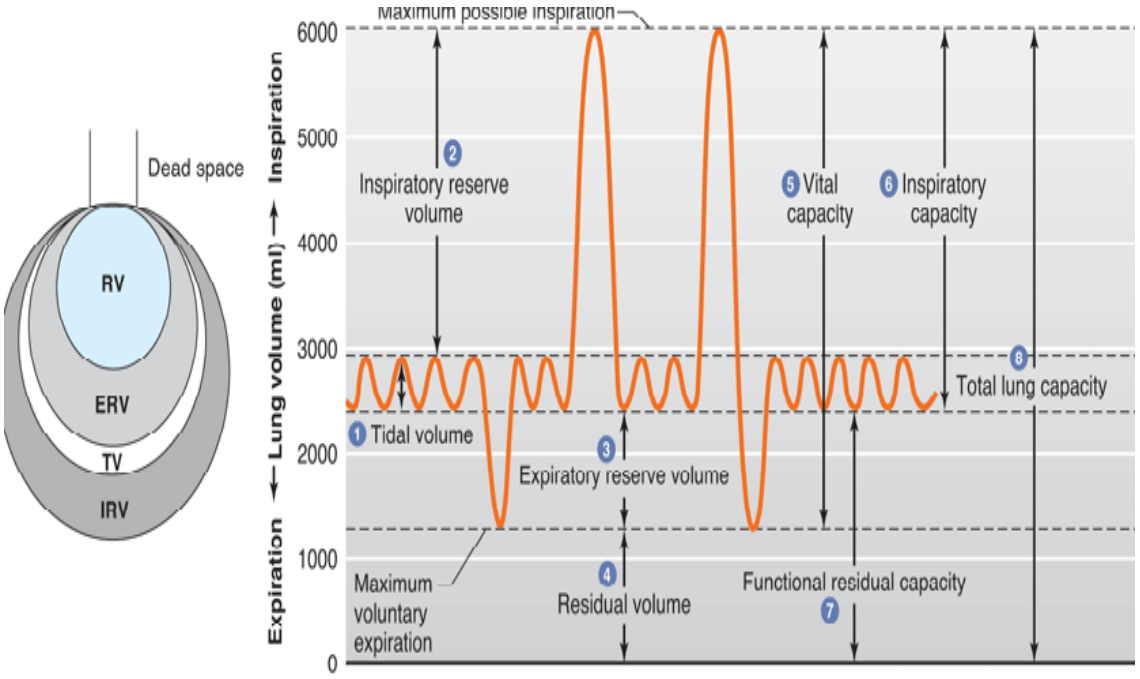

Figure no: 6 Lung volumes and capacities

LUNG INDICES

1. Forced Vital Capacity (FVC)

It is defined as the maximum volume of air expired forcefully and rapidly after a maximal inspiration. Normally FVC equals VC or FVC and VC should be within 200ml of each other. When FVC is < 80% of predicted value it is abnormal. But low FVC is a nonspecific finding. FVC may be low in both obstructive and restrictive disorder. But in restrictive disorder FVC is too low compared to FEV1.

2. Forced Expiratory Volume in one second (FEV 1)

It is the volume of air expired in first second of an FVC maneuver. When it is <80% of predicted value it is considered to be abnormal. It is also a non-specificmeasurement.FEV1 may be low in both obstructive and restrictive disorders, but in obstructive disorder FEV1 is considerably low when compared to FVC.

3. FEV1 / FVC RATIO (OR) FEV1%

The FEV1 expressed as a percentage of VC or FVC. Normal value is 70%. FEV1% = (FEV1/FVC) × 100.

The relationship is a component of most lung function reports.

4. FEF 25-75%

It is expressed as forced expiratory flow over the middle half of the FVC maneuver. It is an indicator of status of medium to small airways. Normal value for healthy young adults is around 4 to 5 liters per second. When it is measured in percentage the normal value is 65% of predicted value.

5. Maximum Voluntary Ventilation (MVV)

It is the maximum volume of air expired in a specific period of time (12 seconds for normal subjects). It tests the overall function of the respiratory system. It is influenced by airway resistance, respiratory muscle, compliance of the lung and chest wall and ventilator control mechanisms. Values in healthy

young men average between 150 – 200 L/min. MVV is decreased in patients with moderate or severe obstructive disease. MVV may be normal in patients who have restrictive pulmonary disease. They can compensate by performing the MVV maneuver with VT and breathing rates.

6. Slow Vital Capacity (SVC)

The volume of gas measured from a slow, complete expiration after a maximal inspiration, without forced or rapid effort is known as vital capacity. It is also referred to as the slow vital capacity, distinguishing it from forced vital capacity.

7. Peak Expiratory Flow (PEF)

The maximal expiratory flow achieved during a maximum forced expiration initiated at TLC. PEF primarily measures large airway function. Effort dependence of PEF makes it a good indicator of patient effort during spirometry. It is particularly useful for monitoring asthma patients at home.[28 &29.]

TYPES OF FUNCTIONAL LUNG IMPAIRMENT

There are two major patterns of abnormal lung function. They are:

1. Restrictive pattern
2. Obstructive pattern.

In restrictive lung function, there is a decrease in lung volume (FVC). The subject inhales reduced volume of air due to reduction in total lung capacity.

Obstructive lung function is indicated by a decrease in the FEV1/FVC ratio. Decline in FEV1/FVC usually results from obstruction in large airways while fall in FEF25-75% signifies small airways obstruction. Asthma and COPD shows Obstructive type of lung function.

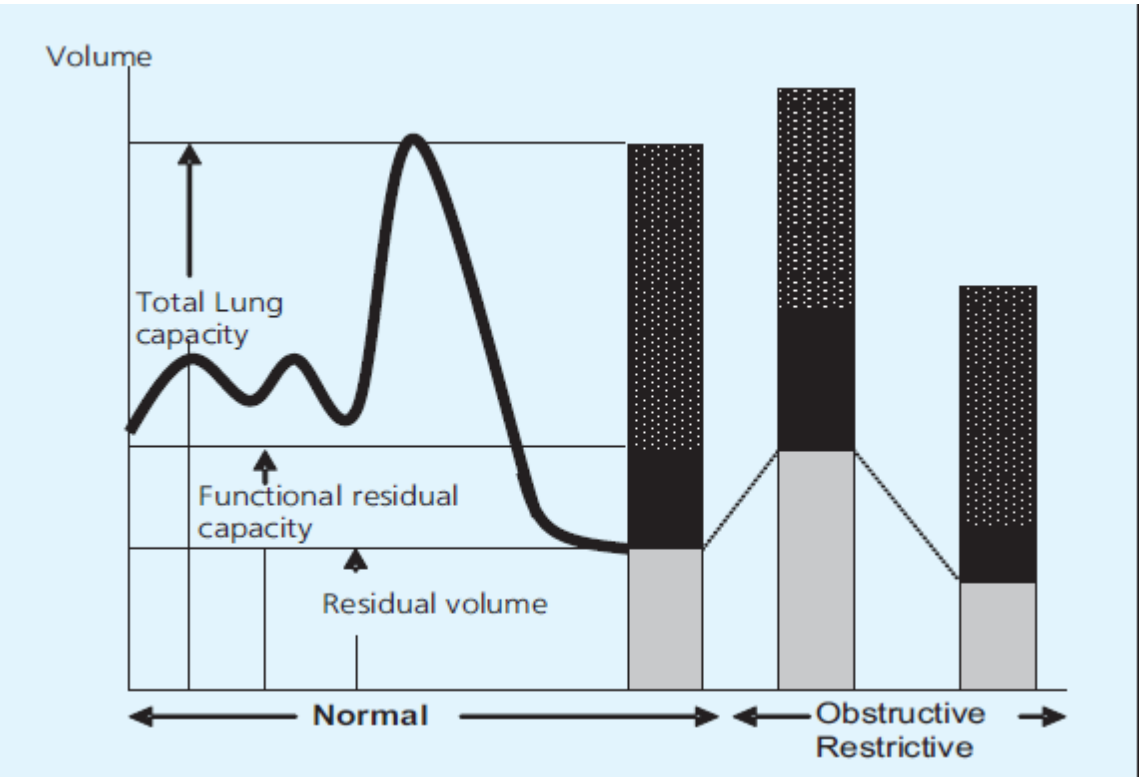

Figure no: 7 Normal lung volumes and changes in lung impairment

AIR FLOW MEASUREMENTS OF OBSTRUCTIVE & RESTRICTIVE DISEASE

In a healthy normal adult male, FVC is approximately 5.0 L, FEV_1 is approximately 4.0 L, and thus, the calculated FEV_1/FVC is 80%. As would be expected, patients with obstructive or restrictive diseases display reduced FVC, on the order of 3.0 L, and this measurement alone does not differentiate between the two. Measurement of FEV_1 can significantly vary between the two diseases.

In obstructive disorders, patients tend to show a slow, steady slope to the FVC, resulting in a small FEV_1, on the order of 1.3 L. However, in the restrictive disorder patients, air flow tends to be fast at first, and then due to the loss of elasticity, quickly levels out to approach FVC. The resultant FEV_1 is much greater, on the order of 2.8 L, even though FVC is equivalent. Obstructive disorders result in a marked decrease in both FVC and FEV_1/FVC, whereas restrictive disorders result in a loss of FVC without loss in FEV_1/FVC.[27]

SPIROMETRY

Spirometry is the most basic and easiest test to measure the pulmonary function parameters and to differentiate lung disorders. However, spirometry must be performed correctly because it may yield a false positive response if performed poorly. Nowadays modern computerized microprocessor based portable spirometers are available to measure pulmonary function parameters.

TYPES OF SPIROMETERS

Based on the principle by which they work the spirometers are of two types.

1. Volume displacement spirometers

The amount of air exhaled or inhaled within a certain time is recorded by such spirometers. The following are widely used volume displacement spirometers.

- Water seal spirometer
- Dry rolling seal spirometer
- Bellows spirometer

2. Flow sensing spirometers (pneumotachometer)

They measure how fast the airflows in or out as the volume of air inhaled or exhaled increases. The common types are:

- Rotating vanes (turbine)
- Pressure differential flow sensing spirometer
- Hot wire anemometers
- Pitot tube flow sensing spirometers
- Ultrasound devices.

INDICATIONS FOR SPIROMETRY

1. To detect presence or absence of lung dysfunction

2. To assess severity of lung disease
3. To monitor the disease progression
4. To assess the efficacy of treatment given
5. To measure the effects of occupational and environmental exposure of airpollutants
6. To assess fitness of patient prior to surgical procedures.
7. To quantify the impairment or disability.[30 &31.]

CONTRAINDICATIONS FOR SPIROMETRY

- Any respiratory infections
- Recent myocardial infarction within 1 month prior to the procedure.
- Unstable cardiovascular status.
- Haemoptysis of any cause.
- Pneumothorax
- Recent surgeries of eye / thorax / abdomen
- Stress incontinence.
- Dementia or confused patient.
- Oral or facial pain exaggerated by the mouth piece. [30 &31.]

RECORDING OF SPIROMETRY

It is recorded both graphically and numerically. Graphically it is recorded as

- Spirogram – volume versus time graph
- Flow rate versus volume – it can be either
- Flow volume curve when only expiratory flow is recorded
- Flow volume loop when both expiratory flow and inspiratory flowis recorded.[32]

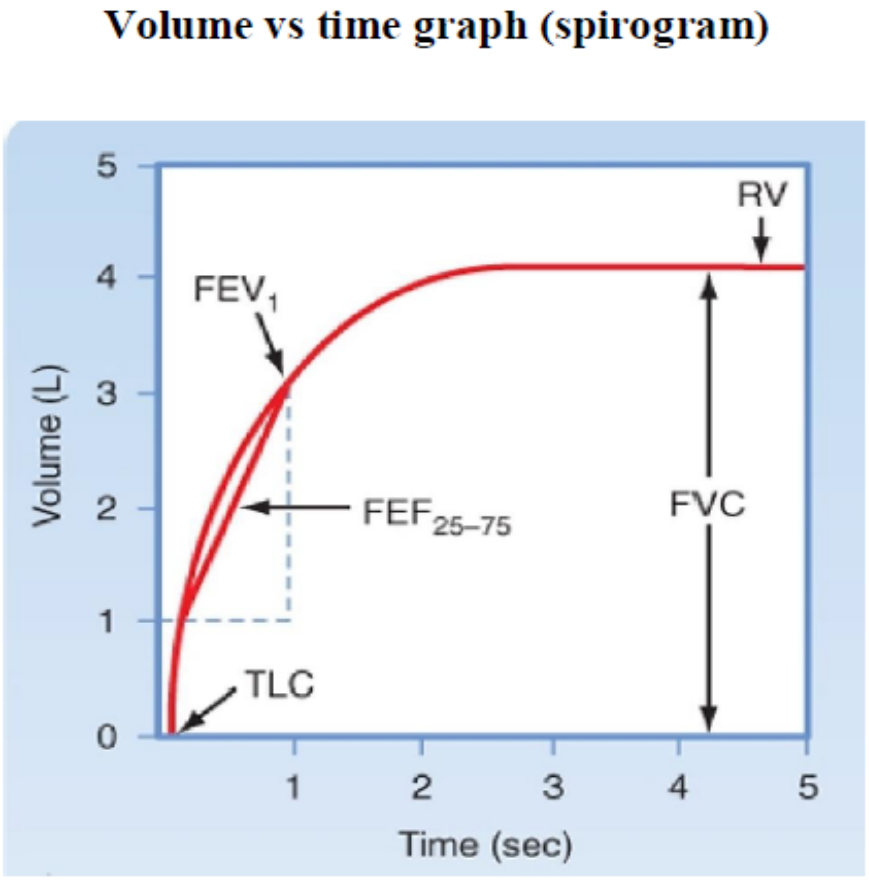

Figure no: 8 Volume vs Time graph (spirogram)

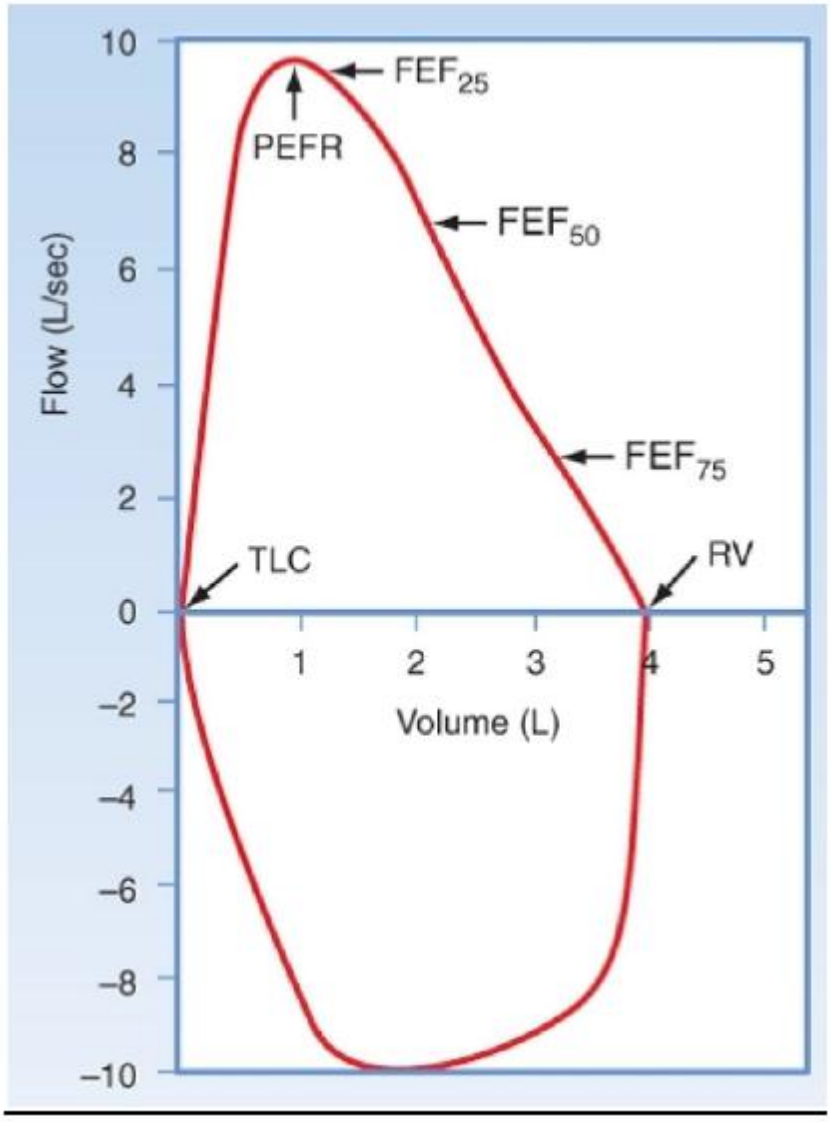

Figure no: 9 Flow – Volume loop

Flow is plotted on the vertical axis and volume is plotted on the horizontal axis.

Expiratory flow is plotted upward and inspiratory flow is plotted downward. Peak flows for expiration and inspiration (PEF and PIF) can be read

directly and the instantaneous flow (FEF) at any point in the FVC also can be measured directly.

LUNG DISORDERS AND ITS PATTERN IN SPIROMETRY RESULTS

Normal value

- FEV1 and FVC >80% predicted value
- FEV1 / FVC ratio >70% of predicted value

Obstructive lung disorders

- FEV1 <80% of predicted value
- FVC normal or reduced (if reduced usually to a lesser degree than FEV1)
- FEV1 / FVC ratio <70% of predicted value

Restrictive lung disorder

- FVC <80% of predicted value
- FEV1 normal or reduced (if reduced usually to a lesserdegree than FVC)
- FEV1 / FVC ratio 70% or >70% of predicted value

Mixed function disorder (Both obstructive and restrictive)

- FVC and FEV1 <80% of predicted value
- FEV1 / FVC ratio <70% of predicted value.[33]

Table no: 1 Interpretation of spirometry data

PFT Parameters	Obstructive Pattern	Restrictive Pattern	Mixed Pattern
FEV1	Reduced	Reduced / Normal	Reduced
FVC	Reduced / Normal	Reduced	Reduced
FEV1 / FVC	Reduced	Normal / Increased	Reduced

Easy on PC Spirometry

REVIEW OF LITERATURE

Air pollution is recognized as a major threat to human health. The United Nations Environment Programme has estimated that globally 1.1 billion people breathe unhealthy air (UNEP, 2002). Epidemiological studies have shown that increased concentrations of ambient air particles are associated with a wide range of effects on human health, especially on the cardio-respiratory system.[4]

Air pollution in Asian cities is closely related to the trends in economic and social development. Besides, rapidly increasing industrialization, urbanization, population growth and demand for transportation along with climatic conditions influence air pollution in many Indian cities. In recent time, India is experiencing a rapid growth and economic development reflected by industrialization, urbanization, rise in income and motor vehicle use. Currently about two-third of Indians live in rural areas, but the pattern is changing rapidly as more number of people are moving towards the cities in search of livelihood.

AIR POLLUTION AND RESPIRATORY DYSFUNCTION

Pulmonary function can be affected by several factors including genetic predisposition, weather, season, time of the day, the basic respiratory health of the subject, smoking status, allergens and air pollution.

Pope et al. 1995; **Dockery et al.**1993[34] had done several population-based studies taken up in the industrialized countries and their investigations confirmed the adverse effects of air pollution on human health. They also did series of

epidemiological studies that were followed in a short period of six years from 1989 to 1995 revealed the role of particulate matter as the chief mediator of toxic effects of air pollution .His finding opened a floodgate of epidemiological and toxicological effects of fine and ultrafine particulates of air pollution on lung function.

Badami et al in the year 2005[35]showed in their study that in the last three decades, the number of motorized vehicles in India has increased 29-times, from 1.9 million in 1971 to 55.0 million in 2001. The increase was not uniform for all vehicle types: it was 7-fold for buses, 9-fold for trucks, 10-fold for car, jeeps and taxis, but a remarkable 67-fold for two-wheelers. In general, the chief contributor to outdoor air pollution is the combustion from these vehicles. In most of the cities with increasing population size and economic activity, the major source of combustion is fuel use that adds to the pollution which reflects on the health of an individual.

Bener et al in 1998[36]in his study showed that there is a high prevalence of chronic cough, and sinusitis has been reported among garage workers and taxi drivers who were exposed to vehicular exhaust in United Arab Emirates.

Basu et al in 2001[37]has done similar studies which were conducted in Kolkata city showed that there is an association with traffic related air pollution and high prevalence of cough, bronchitis and asthma in the city. This is caused by inflammation of the upper airway as the result of infections and may be associated with chronic bronchitis.

PATHO PHYSIOLOGY

More than 40 different cell types have been identified in the normal lung and airway. The major cell types are: basal cell, Intermediate cell, ciliated and non-ciliated columnar epithelial cells, goblet cell, Type I and Type II pneumocytes and alveolar macrophages.

AMs represent the first line of cellular defense in the lung. They play an important role in clearance of particle from the inner airways by phagocytosis, generation of oxygen radicals, and endocytosis of insoluble particles and dust .They also possess potent antimicrobial activities by local release of degradative enzymes and reactive oxygen metabolites. AM is the most secretory cell in the body. They actively participate in inflammation, wound healing and tissue repair through their secretory products. They release inflammatory cytokines like tumor necrosis factor alpha (TNF-α), interleukin-1 (IL-1), chemotactic factors like leukotriene B-4 (LTB4), platelet-activating factor (PAF) and chemotactic cytokine such as interleukin-8 (IL-8) that strengthens the pulmonary defense mechanism.[38]

Both acute and chronic exposures to air pollution have been shown to directly affect the structural integrity of the respiratory system. Continued exposure of chemicals can cause necrosis and subsequent sloughing off of ciliated epithelial cells.

A study by, **Salvi et al.**(1999)[39] showed exposure to diesel exhaust had significant increase in neutrophils, mast cells, CD4+ and CD8+ T-lymphocytes,

along with upregulation of endothelial adhesion molecule, ICAM-1 and VCAM-1 in airway lavage.

Controlled exposure of healthy human volunteers to diesel exhaust particles (DEP) for 1 hour produced marked cellular inflammatory response in the airways involving neutrophils, mast cells and lymphocytes. It has been demonstrated that DEP elicits remarkable increase in neutrophils in the proximal airways, while the lymphocyte and histamine responses were found in the distal airways.[39]

A fibrous protein namely elastin has been widely distributed in the elastic tissues of lung. A proteolytic enzyme found in the lysosomes of neutrophils and alveolar macrophages (AM) is Elastase that is capable of destroying elastin, collagen and fibronectin. Elastase also playsan important role in wound healing and disposal of damaged cells and debris. There is enhanced release of elastase production and secretion by the AM by the inhalation of non-degradable irritant materials from the atmosphere.[40] Release of excess elastase from neutrophils and activated AM promotes the development of emphysema as it leads to degradation of alveolar wall.

A pioneering study by **Mylius and Gullvag**(1986)[41] demonstrated several fold increase in the number of AM in sputum of persons exposed to industrial air pollution. The AM count was found to rise with increasing level of particulate pollution in the workplace. They advocated the number of AM as a reflection of the reaction of lung to air pollution.

Lundborg et al., 2001 and **Moller et al.,** 2002[42]in their study showed ingestion of ultrafine particles by the AM may lead to impairment of phagocytic activity of these cells, particularly after infection that induce an increased production of Interferon gamma. The macrophages with their help of cytoskeletons are important for migration, phagocytosis of foreign materials and intra cellular transport and digestion. Urban dust and diesel exhaust contains ultrafine particlesthat have been shown to cause cytoskeletal toxicity which leads to impaired function of the macrophages that causes lung defects.

PULMONARY FUNCTION TEST

Definition:

Pulmonary function tests are a group of procedures that measure the function of the lungs that reveals the problems in the way a patient breathes. Pulmonary function tests helps to diagnose a wide variety of respiratory diseases which might not otherwise be obvious to the doctor or the patient. These tests are important because many kinds of lung problems if detected early can be treated successfully.

History:

- Hippocrates (460-377BC) and Galen believed that breathing is for cooling of the heart.
- Galen (130-211AD) gave idea the respiratory act is brought about by the diaphragmatic contraction and chest wall movements.

- In 1680 G A Borlley measured the inspiratory volumes for the first time and he also mentioned about the Residual Volume.[43]
- During 17th century they used mercurial air holding machine and hydrogen dilution techniques were used to measure Residual Volume.
- Then in 1800, Sir Humphrey Davy measured the lung volumesby using Hydrogen.
- In 1813, Kentish E used a simple "Pulmometer" to study theeffect of diseases on lung volumes. He used an inverted graduated bell jar with an outlet standing in water and the top of the bell jar is controlled by a tap. The volume of airwas measured in units of pints.[43]
- In 1831, Thrackrah C.T described the pulmometer similar to that of Kentish.He formulated the device as a bell jar with an opening for the air to enter from below.There was no correction for pressure. Therefore, the spirometer was not only used to measure the respiratory volume, but also the strength of the respiratory muscles.
- In 1845, Vierordt in his book illustrated accurately the measurement of volume of expiration. However, he also completed accurate measures of other volume parameters by using his "Expirator". The residual volume and vital capacity parameters which were described by him are used today.
- In 1846, John Hutchison measured Vital Capacity and described the subdivisions of lung volumes. In 1880 N Grehnet measured the Functional Residual Capacity (FRC).

- In 1893 Hermansen, introduced Maximum Breathing Capacity. Thesame test now is called Maximum Voluntary Ventilation.
- In 1902, Brodie T.G wasthe first person to use a dry-bellowed wedge spirometer.
- In 1950, the unanimously agreed nomenclature was given by 'U.S.A.Respiratory Physiologists committee'.
- Titteneau and Pinell and also Gaensler in the year 1951 developed the technique for measuring Timed Volume. The procedure was referred to 'Forced Vital Capacity Maneuver' which quantifies the air volume dynamics and describes the rates of air flow along the respiratory tree which was useful for obtaining pulmonary function tests.[44]
- Determination of average airflow during middle half of FVC i.e.FEF 25%-75% was described in the year 1955. Wright B.M. and McKerrow C.B. first introduced the peak flow meter in 1959.
- In 1969, DuBois A.B. and Van de Woestijne K.P. had done experimentson humans the whole body plethysmograph.
- In 1974, Campbell et al. refined the peak flow meter which was previously used and put forward a cheaper and lighter version of a peakflow meter.[44]
- Currently, there are many techniques which were used for assessing both their individual components and integrated performance of respiratory system.

A prototype of the water sealed modern Spirometer was developed. A treatise on the capacity of the lungs and on respiratory functions also defined the functional subdivisions of lung volume. In more than 1800“healthy cases”,the ‘Vital Capacity’ (VC) was measured and these values were related to the age, height and weight of the subjects and thereby establish basis for determining normal values.[45]

Recording of Timed Vital Capacity was made in the year 1951 and in 1955 the measurement of the maximal mid expiratory flow was done. With the introduction of a cathode ray oscilloscope and pressure sensitive transducer which was capable of reproducing rapidly changing signals permitted to measure lung volumes and airway resistance.

In the mid 1960`s computers were used to display analysis of pressure and flow volumes as three dimensional graphs. They applied these techniques for the analysis of forced expiratory maneuver.

The wide spread application of microprocessors began in the late 1970`s which gave way to the sophisticated pulmonary testing applications. The wide variety of flowsensing Spirometers, metabolic measurement systems and pulse oximeters are few examples of many techniques which has been used for assessing the integrated performance of respiratory system and its individual components.

Changes in lungvolume with inspiration and expiration and the absolute volume of air that the lung can hold at various phases of respiratory cycle are the important quantitative aspects of respiratory functions.

The total volume of gas in the lungs is conventionally sub-divided into compartments as volumes and combination of two or more volumes as capacities has been made for the purpose of quantification and comparison of the values.[45]

Spirometeris an instrument used for the measurement of lung volumes and capacities. The facilities to perform PFT were rather limited at Indian centers until 1990. Of all the different PFT's, spirometry is a most widely used investigation in routine clinical practice.[46]

Spirometry is derived from the Greco-Latin term which means "to measure breathing".Undoubtedly, Spirometry is a useful investigative tool in the hands of physicians, surgeons, anesthetists and to all others who deal with respiratory and cardiovascular problems.[47]

Spirometry is useful to differentiate an obstructive, restrictive or a mixed defect in the lung.Respiratory diseases are classified under one of these groups based on the measurement of lung volumes (VC) and flows (FEV1, FEV1/FVC). A variety of factors influences these above measurements which must be considered carefully before one is evaluating these values.

Spirometers are available in a variety of configurations a spirometer, including the waterless and rolling seal type are available. Stead-Wells waterseal type is an instrument that directly measures the volume of air displaced ormeasures airflow by a flow-sensing device, such as a pneumotachometer or a tube containing a fixed resistance to flow.

Some Spirometers primarily sense volume and are known as Volume type Spirometer, while others primarily sense air flow at particular times, which are known as Flow type Spirometers.

Volume spirometers record the forced expiratory maneuver as it is produced. When the subject breathes into a mouthpiece, the air moves a cylinder, a plastic bell, or a rubber or plastic diaphragm, which in turn moves a pen that traces a curve on a moving paper graph. The water seal, dry rolling seal, and bellows spirometers are the three most widely used types of volume spirometers.

Flow type Spirometer uses pneumotachograph or rotating turbines to determine air flow. Flow spirometers measure how quickly air flows past a detector and then derive the volume by electronic means. They record the flow rate at very brief intervals, such as 30-300 times a second, and use the data obtained to reconstruct the flow rate at each point in time and volume. This process is called digitization. The most common types of flow spirometers are the pneumotachographs, hot wire anemometers, and rotating vanes.

There are two types:

1. Hot wire
2. Flow resistive

In hot wire type, air flowing past a heated wire cools the wire, thereby altering its resistance in proportion to changes in airflow. Flow resistive pneumotachograph contains a resistive element composed of parallel tubes, a

wire mesh or a fibrous paper like element. Airflow through resistive element results in pressure gradient across the device, which can be measured by a very sensitive differential pressure gauge.

The drop in pressure across the resistive element is sensed by a pressure transducer and this pressure is converted to a voltage output that is proportional to flow. Moreover, the flow signal can be integrated electronically to yield volume.

With an electronic Spirometer, all the parameters pertaining to lung volumes and flow rates can be measured just by demonstrable maneuvers.[48]

EFFECT OF VEHICLE EXHAUST ON PULMONARY FUNCTION TESTS

Taggartet al[49] and **Rusas et al**[50]in their study concluded that, exposure to air pollutants is known to be harmful to health, in general, and to the lungs in particular. In this respect, traffic police personnel, due to the nature of their job, are at a particular risk, since they are continuously exposed to emissions from vehicles. These personnel have to undergo physical strain in an environment polluted by fumes, exhaust of vehicles, use of blowing horns, blow of dust in the air by a speeding vehicle, etc. Above study was conducted among traffic personnel who were also subjected to prolonged exposure to dust resulting in respiratory problems.

Similar study was conducted by **Vinayet al**[51] (2016) among traffic policemen in Gorakpur city Uttar Pradesh. Pulmonary function tests (PFTs) were performed with computerized spirometer. Comparison of ratio FEV1/FVC among two populations revealed lower values for traffic policeman group. They showed an airway obstructive pattern of disease in their study, which was lower in Traffic policemen than control group and this was significant in non-smokers groups not in smokers groups between control and traffic policemen because smoking itself affects lung functions. The respiratory parameters were altered among policemen due to traffic related air pollution,being a major risk factor in the development of respiratory obstruction among exposed groups.

Singhalet al[52]carried out a study on petrol pump workers in Delhi. His study was done to assess the pulmonary functions in petrol pump workers who are continuously exposed to petrol/diesel vapors during duty hours. 30 healthy non-smoker males working in petrol pump for more than one year formed the study group, while 30 healthy non-smoker males from hospital staff served as control group. The pulmonary functions were assessed using computerized spirometer. The FVC and FEV1 were decreased in the study group while their ratio did not differ much. Both the inspiratory and expiratory flow rates were also decreased in the study group affecting lower airways with restrictive pattern of disease.Occupational exposures to petrol/diesel vapors have been shown to affect functioning of different systems of the body.

Ajayet al[53] in his study compared the lung function of autorickshaw drivers in terms Peak Expiratory Flow Rate (PEFR) with the residents of Urban Davangere. Healthy adult subjects of urban residential areas of Davangere (control) and healthy nonsmoking auto rickshaw drivers were selected randomly from the population of Davangere. Wrights Peak flow meter which is a portable device for measuring ventilator functions. Comparisons were done between 2 groups and there has been a statistically significant decrease both in actual PEFR and percentage of predicted PEFR among the individual groups classified on basis of duration of driving. From the present study it was concluded that espiratory functions of auto rickshaw drivers who are continuously exposed to emissions from vehicles were significantly reduced. Long term exposure to the air pollutants leads to deleterious effects on the respiratory functions of automobile drivers.

Iraniet al[54]in his study assessed respiratory health in auto rickshaw drivers of Aurangabad, forming study group and non-smoker employees of the college formed control group in age group of 25- 45 years. They found that there was significant decline in lung functions in auto rickshaw drivers as compare to control group.It was observed that FVC and FEV1 in auto drivers though less than control group but was in normal limits. FEV1% and FEF 25-75 of auto drivers was significantly less than control group which exhibit a significant decline in lung functions. Duration of auto driving had no relation with decrease in lung functions as duration considered in study was less than 10 years. This

concludes that there is adverse effect of vehicle exhaust on lung functions leading to obstructive type of lung disease.

Similar study by **Anujet al**[55] (2008) using Spirometry was done. They studied the lung functions in petrol station workers compared to healthy controls in Pune city to assess the long term effects of work environment on lung function. The workers were divided into 5 groups for analysis of data based on the number of years of work in the petrol pumps. Outdoor air analysis was also carried out. The FVC, FEV1and PEF declined significantly with increasing years of work in petrol stations in both smokers and non-smokers.

Abdulbariet al[56](1998)also does similar study to determine the prevalence of some respiratory symptoms among occupationally-exposed garage workers and taxi drivers. This study involved garage workers and taxi drivers, matched for age, sex, nationality and duration of employment in UAE. The data on chronic respiratory symptoms showed that garage workers had higher prevalence of symptoms than taxi drivers, being significantly greater for chronic phlegm, dyspnea and sinusitis. Almost all forced spirometric tests in the exposed garage workers were lower than in taxi drivers. They concluded that a high prevalence of respiratory symptoms is associated with exposure to motor vehicle exhaust emission in garage working places. Long-term working as garage workers in the United Arab Emirates (UAE) may be associated with the development of chronic respiratory symptoms.

Zuskinet al[57] (1994) in his study evaluated acute and chronic respiratory symptoms as well as ventilatory capacity in bus drivers and mechanics. Bus drivers and mechanics demonstrated a significantly higher prevalence of most chronic respiratory symptoms when compared to control workers. Bus drivers and mechanics who were smokers had significantly higher prevalence of respiratory symptoms than nonsmoking bus drivers. Bus drivers and mechanics employed for more than 10 years also exhibited higher frequencies of respiratory symptoms than those exposed for 10 years or less. The ventilatory capacity data demonstrated lower values for all parameters, particularly FEF25, compared to control worker values as well as to predicted normal values, for bus drivers and mechanics who were smokers. Their data showed that long-term employment in the transport industry of bus drivers and mechanics, particularly in combination with smoking, may be associated with the development of chronic respiratory symptoms and lung function impairment.

Ibrahimetal[58] conducted study in non-smoking auto rickshaw drivers in Kerala and compared with controls. All the lung function parameters were reduced significantly in the auto rickshaw drivers as compared to control subjects in the same age group and socio economic status. He also found that respiratory function tests of auto rickshaw drivers who had worked for more than 10 years were more affected than those who had worked for less than 10 years. Majority of their subjects were found to have mixed obstructive and restrictive lung impairment.

Gavaliet al[59] (2015) in his study evaluated only FVC in auto rickshaw drivers of Pune city among with normal citizens and he found restrictive type of lung impairment among auto drivers when compared with control group. He further evaluated those drivers who are driving for more than 5 years very highly significant reduction in their FVC functions. In their study they had not compared the other parameters between the groups.

Similar study conducted by **Bijendraet al**[60] among diesel taxi drivers of Bikaner city and compared it with healthy medicos. Restrictive impairment was found in 87% of study group, of which 50% were smokers and 37% were nonsmokers, mixed pattern in only 13% of study group of which 7% were smokers & 5% nonsmokers. He concluded to be indicative of mixed pattern lung impairment.

Another study by **Purushottamet al**[61] in auto taxi drivers at Hooghly and compared it with healthy males residing in same geographical area. Restrictive type of impairment was found in smokers and non-smokers study subjects. Obstructive and mixed types of impairment were noted in smokers study subjects. There was higher percentage of respiratory impairment in smoker drivers than non-smoker drivers who were exposed to petrol engine emission from auto taxi.

Similar study by **Afshanet al**[62] assessed PFT in auto rickshaw drivers of Gulbarga city. He compared 50 male auto rickshaw drivers driving for more than 5 years with 50 males of same age group who were not auto drivers. There was a

highly significant decrease in FVC and FEV1 in the study group compared to control group. He found the restrictive pattern of lung impairment among study group, which in turn shows adverse effects of vehicle exhaust on lung functions, mainly on lower airways with restrictive pattern of disease.

Rajkumaretal[63] (1999) in his study also found that (19%) drivers showed normal Pulmonary Function Test (PFT). Eighty percent showed mild and moderate to severe obstruction, of which 48% were nonsmokers and 52% were smokers and the result concludes that auto rickshaw drivers have a high respiratory morbidity due to exposure to pollution.

This study was done to evaluate the involvement of lung structures in auto drivers. Literature highlights the fact that respiratory system is definitely involved in auto drivers exposed to vehicle exhaust but the extent and pattern of lung involvement with respect to duration of their work was inconclusive. So the aim of the present study is to evaluate type of respiratory impairment and its correlation with duration of work in auto drivers.

AIM AND OBJECTIVES

AIM

To evaluate the respiratory impairment by pulmonary function test in auto drivers.

OBJECTIVES

1. To compare the pulmonary functions of auto drivers with general population.
2. To evaluate type of respiratory impairment by pulmonary function test among auto drivers.
3. To assess the severity of impairment with respect to the duration of driving

MATERIALS AND METHODS

STUDY DESIGN - Cross-sectional study

PERIOD OF STUDY - 2016 to 2017

PLACE OF STUDY - Department of Physiology
Chengalpattu Medical College, Chengalpattu.

SELECTION OF SUBJECTS

- 50 auto drivers who were nonsmokers as study group
- 50 normal healthy male of general population who were non smokers as control group
- Detailed history relevant to the study is taken
- Patients who are willing and satisfying the inclusion criteria are to be selected
- Informed consent was obtained from all the patients taken up for study.

INCLUSION CRITERIA

Fifty nonsmoker male auto drivers in the age group of 20–50 years who were driving for more than 2 years are selected as participants.

EXCLUSION CRITERIA

1. Gross pulmonary disease
2. Anatomical deformity of chest & spine
3. Infective lung disease like tuberculosis
4. Severe respiratory distress
5. Cardiac patients
6. Connective tissue disease
7. Post thoracic surgery

EASY ON PC SPIROMETRY

Working principle is the Ultrasound flow sensor measures the transit time which allows the accurate determination of flow velocity independent of temperature, humidity and molar mass of the gas. Since the measuring principle is based on a digital measurement technique the sensor requires only one single calibration and does not change during the sensor's lifetime.

PROCEDURE

- Anthropometric measurements - Height and weight of the subjects were measured.
- Informed consent was obtained.

ON THE DAY OF RECORDING

Subjects were advised to avoid

- Full meals 2 hours prior to the test
- Alcohol consumption 4 hours prior to the test
- Short acting bronchodilators 6 hours prior to the test
- Long acting bronchodilators 12 hours prior to the test
- Use of mobile phone.

SPIROGRAM

FLOW VOLUME LOOP

- Ask the subject to sit comfortably and relaxed in an armed chair with straight back
- Procedure demonstrated to the subject
- Subject is asked to inhale atmospheric air deeply
- Nose clip is placed immediately
- Spirette is kept inside the mouth with lips tightly sealed around it.
- The subject is asked to blow out air as fast and as hard as possible, blast out for a minimum of 6 seconds
- Then immediately ask the subject to inhale deeply with the spirette still inside the mouth (to form a loop)
- Minimum of 3 trials done with an interval of 5 minutes between each trial.
- Best of 3 trials taken for analysis.[64]

NUMBER OF TRIALS

A minimum of 3 acceptable FVC maneuvers should be performed. If a subject is unable to perform a single acceptable maneuver after 8 attempts, testing may be discontinued for that session.

ACCEPTABILITY

1. A good 'start of test' includes
   - An extrapolated volume of 0.2 L less or equal to 5% of the FVC or 150 ml, whichever is greater
   - No hesitation or false start

   A rapid start to rise time.
2. No cough, especially during the first second of the maneuver.
3. No early termination of exhalation.
4. No evidence of leak.
5. A minimum exhalation time of 6 seconds is recommended, unless there is an obvious plateau of reasonable duration for at least 2 seconds or a volume of 40 ml towards the end of the effort.
6. Nose clip may or may not be used while making the FVC effort.
7. Test at ambient temperature 17°C to 40°C, with Spirometer ≥ 23°C, if possible.

REPRODUCIBILITY

1. The two largest FVC`s from acceptable maneuvers should not vary by more than 0.2L.
2. The two largest FEV1 from acceptable maneuvers should not vary by more than 0.2L.[65 &66.]

Table no : 2 INTERPRETATION OF SPIROMETRY VALUES

PFT Parameters	Restrictive	Obstructive
FVC	< 80% of predicted	Normal < 80% of predicted
FEV1	Normal < 80% of predicted	< 80% of predicted
FEV1/FVC	≥ predicted	< predicted

The values (FVC, FEV1 and FEV1/FVC) were compared with average predicted for a subject on the basis of age, sex, built and race.[65] In both the groups, subjects were highly motivated and cooperative. They performed the tests with care and maximal efforts.

SEVERITY OF RESTRICTIVE PATERN

(BASED ON FVC %)

- Mild restriction – 60 to 80%

- Moderate restriction – 45 to 59%
- Severe restriction - <45% [66 &67.]

ANALYSIS

- Percentage of the Predicted values of FEV1, FVC, FEV1 / FVC, FEF25% -75% were taken for analysis.
- The pattern of lung function impairment was assessed from spirometry results using percentage of the predicted values of FEV1/FVC, FVC.
- Severity of restrictive impairment of lung function was assessed using percentage of predicted values of FVC.[67]
- Data was entered in Microsoft Excel spreadsheet and analysis was done in SPSS Version 21.
- Statistical tests like Independent sample 't' test, One way Anova and Correlation was applied.

Recording with Easy on PC Spirometry

RESULTS

A total number of 100 participants took part in the study. Out of 100 participants 50 were forming the study group who were auto drivers and the remaining were normal subjects forming the control group. The study groups were divided into 3 subgroups as 2-10 years, 11-19 years and 20-30 years, based on the duration of driving.

The anthropometric and lung function parameters were processed and analyzed by SPSS version 21. The arithmetic mean and standard deviation was calculated. The mean values of pulmonary function parameters of auto drivers were compared with healthy controls and their significance were derived using Independent sample ‘t’ test.

The pulmonary function parameters were compared with the duration of driving among subjects by using Anova test and Correlation done using Pearson’s correlation coefficient test.

Table no : 3 Descriptive statistics of anthropometric parameters

Anthropometric parameters	Controls (N = 50)				Subjects (N = 50)			
	Min.	**Max.**	**Mean**	**S.D.**	**Min.**	**Max.**	**Mean**	**S.D.**
Age (years)	23	49	37.52	7.4	23	50	39.18	8.3
Height (cms)	151	184	170.52	7.4	156	182	168.72	6.3
Weight (kgs)	44	98	66.44	11.6	55	100	73.70	11.8
BMI (kg/m^2)	16.33	30.70	22.83	3.59	17.96	33.65	25.85	3.59

The above table shows the baseline data of study and control groups. The arithmetic mean and standard deviation of anthropometric measurements of study and control groups were calculated.

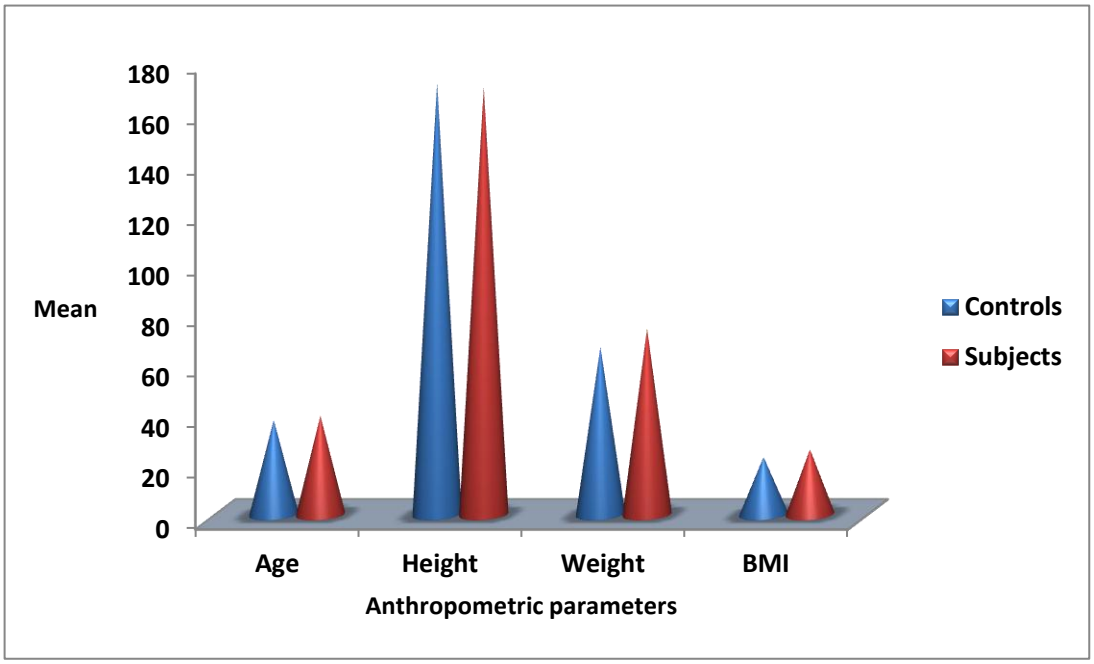

Figure no : 10 Distribution of anthropometric parameters of controls and subjects

It is observed from this figure that, the mean of age, height, weight and body mass indices of both controls and subjects.

Table no : 4 Pulmonary function parameters of controls and subjects

Pulmonary function parameters	Controls (N = 50)				Subjects (N = 50)			
	Min.	**Max.**	**Mean**	**S.D.**	**Min.**	**Max.**	**Mean**	**S.D.**
FVC	51	119	94.72	16.6	47	103	71.40	13.6
FEV1	54	117	91.04	13.8	49	99	73.62	12.9
FEV1/FVC	87	109	96.34	96.3	90	124	105.02	8.1
FEF 25-75%	55	140	102.00	25.5	8	135	93.36	29.9
PEF	55	146	109.28	19.4	55	139	89.52	17.4

The above table shows the pulmonary function parameters of study and control groups. The arithmetic mean and standard deviation of lung function parameters of study and control groups were calculated.

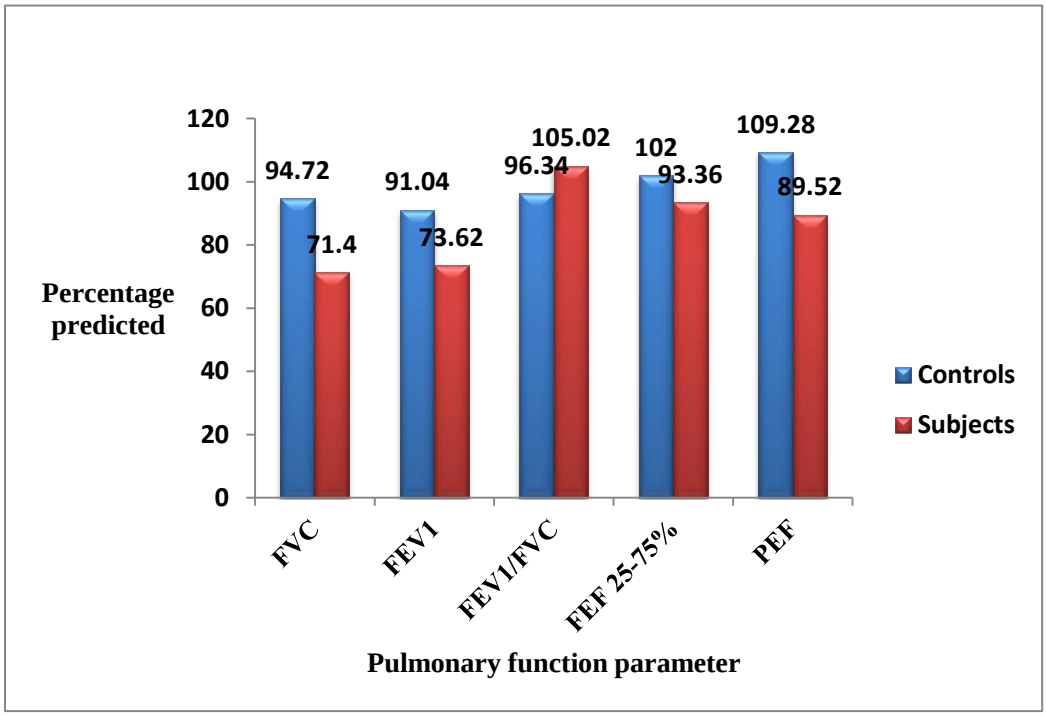

Figure no : 11 Bar chart showing descriptive of pulmonary function parameters

The above figure is the bar chart showing pulmonary function parameters of controls and subject. The percentage predicted values of FVC and FEV1 of study subjects were reduced when compared to controls.

Table no : 5 Comparison of pulmonary function parameters between control and subjects

Pulmonary function parameters	Controls (N = 50)		Subjects (N = 50)		Mean difference	't' value	p value
	Mean	S.D.	Mean	S.D.			
FVC	94.72	16.6	71.40	13.6	23.32	7.685	0.000*
FEV1	91.04	13.8	73.62	12.9	17.42	6.507	0.000*
FEV1/FVC	96.34	5.8	105.02	8.1	8.68	6.136	0.000*
FEF 25-75%	102.00	25.5	93.36	29.9	8.64	1.554	0.123

($p < 0.05$ is considered statistically significant)

The mean (±SD) of pulmonary function parameters of the control group and study group were compared. It was found to be statistically highly significant (P=0.000) for FVC, FEV1 andFEV1/FVC between the study and control groups. The mean values of FVC and FEV1are found to be reduced among subjects when compared to controls and are statistically significant.

Table no : 6 Comparison of pulmonary function parameters with duration of driving among subjects

Pulmonary function parameters	Duration of driving (years)						'F'	p value
	2 -10		11 – 19		20 – 30			
	Mean	S.D.	Mean	S.D.	Mean	S.D.		
FVC	85.00	12.8	71.89	6.5	61.06	10.8	21.55	0.000*
FEV1	85.23	10.3	74.24	9.4	64.56	11.1	15.36	0.000*
FEV1/FVC	102.62	8.8	104.32	7.5	107.5	8.0	1.51	0.231
FEF 25-75%	101.00	25.2	94.32	30.5	86.83	32.5	0.86	0.431
PEF	93.62	16.8	88.26	15.0	87.89	20.3	0.481	0.621

($p < 0.05$ is considered statistically significant)

The mean of lung function parameters with duration of driving shows there is statistically highly significant association between FVC and FEV1 with respect to duration of driving.

Table no : 7 Association between pulmonary function parameter FVC and duration of driving

FVC					
	Sum of Squares	**df**	**Mean Square**	**F**	**Sig.**
Between Groups	4335.266	2	2167.633	21.554	0.000
Within Groups	4726.734	47	100.569		
Total	9062.000	49			

($p < 0.05$ is considered statistically significant)

This table shows that there is statistically highly significant association between FVC and duration of driving among subjects.

Table no : 8 Association between pulmonary function parameter FEV1 and duration of driving

FEV1					
	Sum of Squares	**Df**	**Mean Square**	**F**	**Sig.**
Between Groups	3239.344	2	1619.672	15.359	0.000
Within Groups	4956.436	47	105.456		
Total	8195.780	49			

($p < 0.05$ is considered statistically significant)

This table shows that there is statistically highly significant association between FEV1 and duration of driving among subjects.

Table no : 9 Association between pulmonary function parameter FEV1/FVC and duration of driving

FEV1/FVC					
	Sum of Squares	**Df**	**Mean Square**	**F**	**Sig.**
Between Groups	195.298	2	97.649	1.514	0.231
Within Groups	3031.682	47	64.504		
Total	3226.980	49			

($p < 0.05$ is considered statistically significant)

It is observed from this table that there is no statistically significant association between FEV1/FVC and duration of driving among subjects.

Table no : 10 Association between pulmonary function parameter FEF 25-75% and duration of driving

FEF25-75%					
	Sum of Squares	**Df**	**Mean Square**	**F**	**Sig.**
Between Groups	1542.915	2	771.457	0.857	0.431
Within Groups	42296.605	47	899.928		
Total	43839.520	49			

($p < 0.05$ is considered statistically significant)

It is observed from this table that there is no statistically significant association between FEF 25-75% and duration of driving among subjects.

Table no : 11 Association between pulmonary function parameter PEF and duration of driving

PEF					
	Sum of Squares	**Df**	**Mean Square**	**F**	**Sig.**
Between Groups	295.941	2	147.971	0.481	0.621
Within Groups	14464.539	47	307.756		
Total	14760.480	49			

($p < 0.05$ is considered statistically significant)

It is observed from this table that there is no statistically significant association between PEF and duration of driving among subjects.

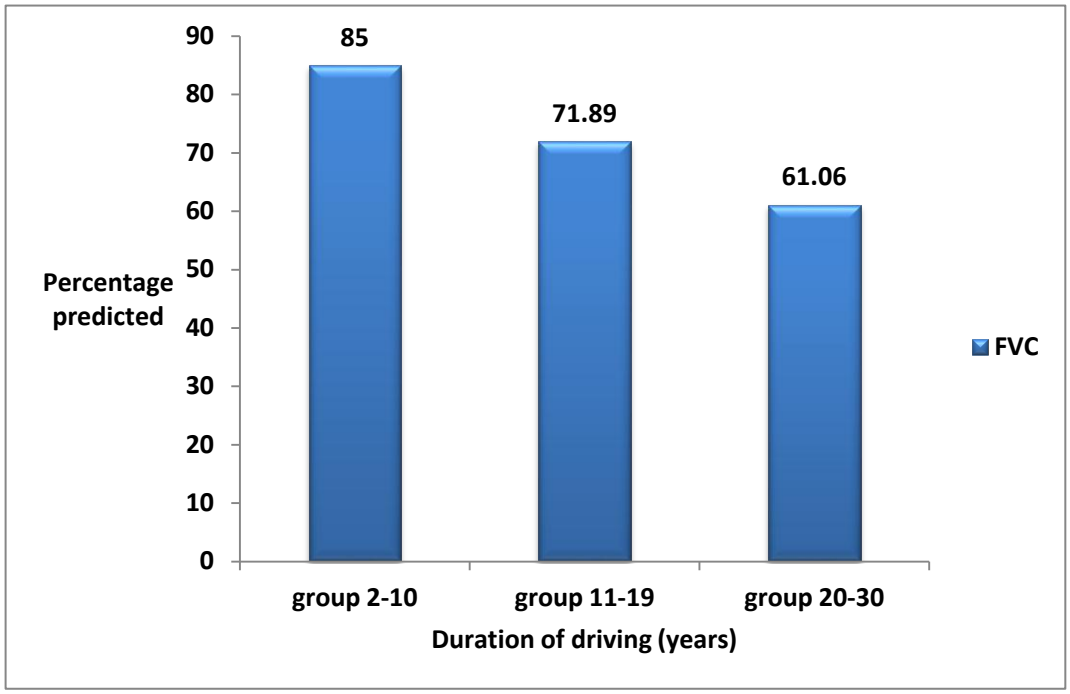

Figure no : 12 Bar chart showing FVC of subjects according to duration of driving

This above picture is the bar chart showing the distribution of pulmonary function parameter FVC of subjects according to duration of driving. As the duration of driving increases the percentage predicted FVC decreases.

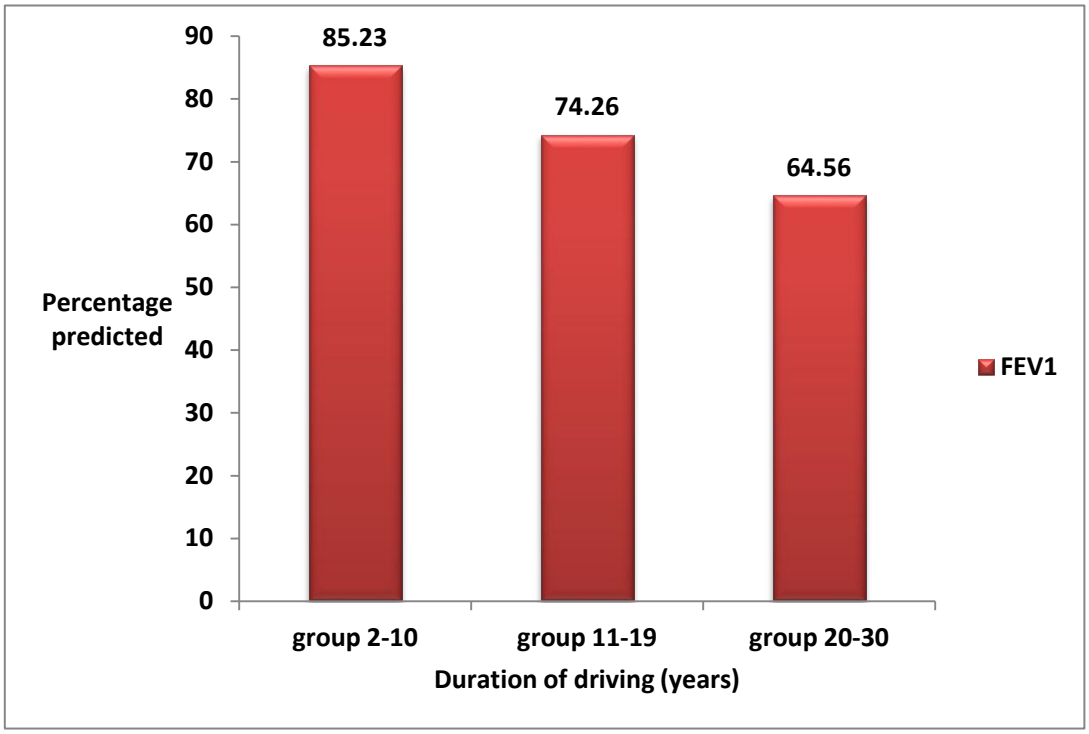

Figure no : 13 Bar chart showing FEV1 of subjects according to duration of driving

This above picture is the bar chart showing the distribution of pulmonary function parameter FEV1 of subjects according to duration of driving. As the duration of driving increases percentage predicted value of FEV1 decreases significantly.

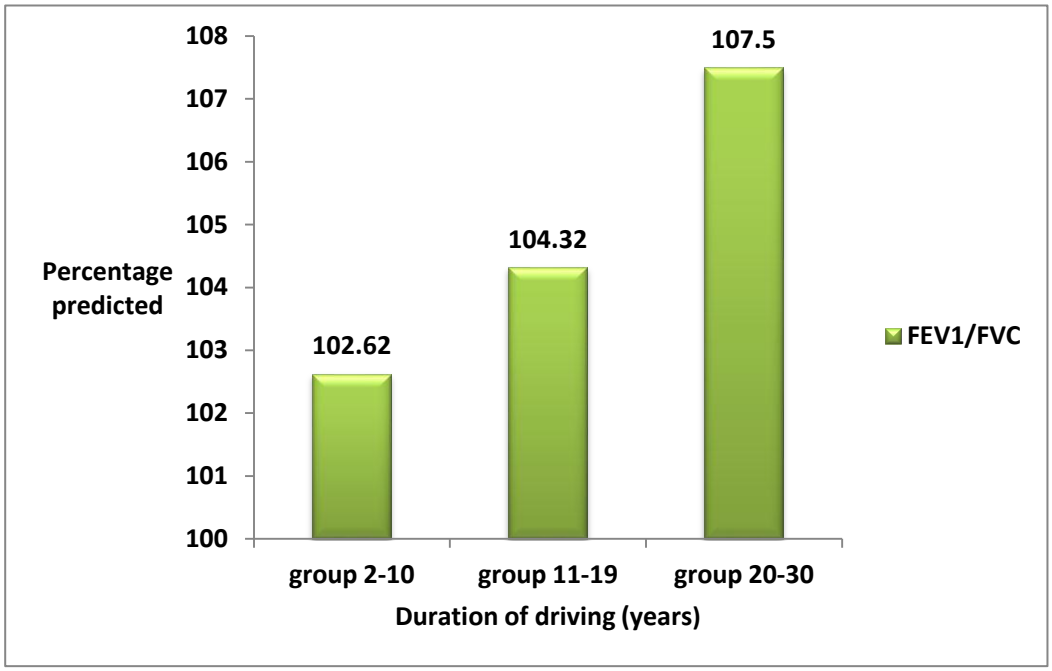

Figure no : 14 Bar chart showing FEV1/FVC of subjects according to duration of driving

This above picture is the bar chart showing the distribution of pulmonary function parameter FEV1/FVC of subjects according to duration of driving. As the duration of driving increases the percentage predicted value of FEV1/FVC increases significantly.

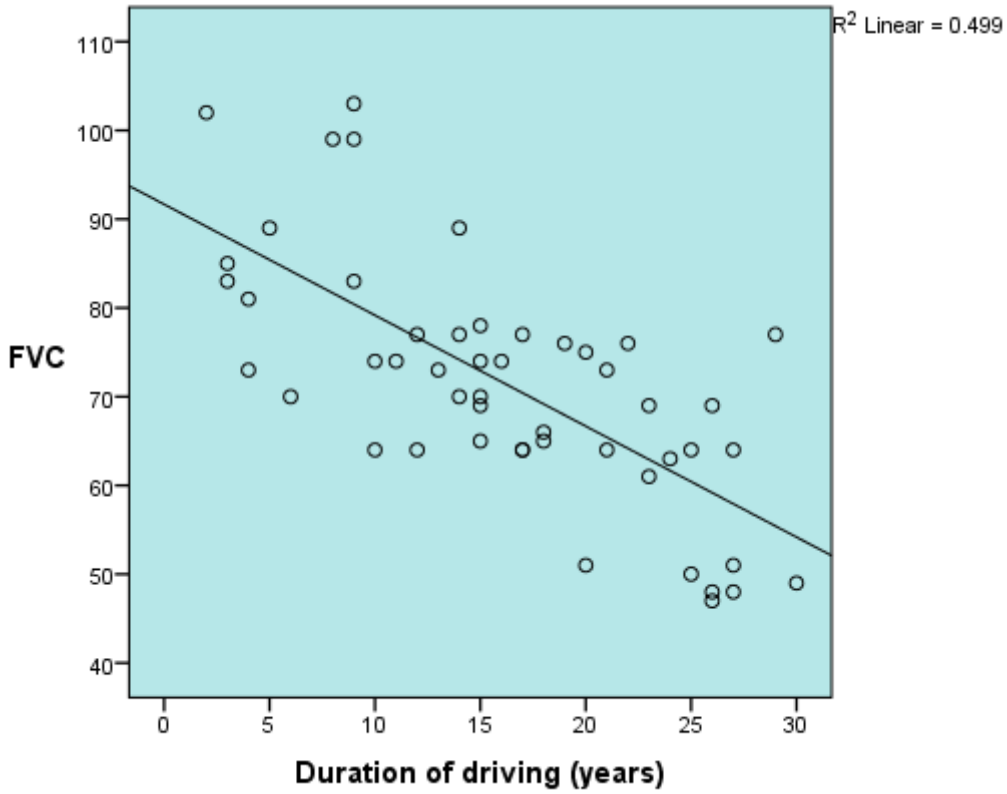

Figure no : 15 Scatter plot showing the relation between pulmonary function parameter FVC of subjects and duration of driving

It was observed from the scatter plot shown above that the pulmonary function parameter FVC of subjects was having a negative relationship with the duration of driving.

Table no : 12 Correlation between pulmonary function parameter FVC of subjects and duration of driving

Correlation between FVC and duration of driving			
		FVC	**Duration of driving**
FVC	Pearson Correlation	1	-0.707**
	Sig. (2-tailed)		0.000
	Total	50	50
Duration of driving	Pearson Correlation	-0.707**	1
	Sig. (2-tailed)	0.000	
	Total	50	50
**. Correlation is significant at the 0.01 level (2-tailed).			

Pearson correlation shows that there was statistically highly significant negative relationship between pulmonary function parameter FVC of subjects and duration of driving ($r = -0.71$, $p < 0.01$), i.e. as the duration of driving increases FVC of subjects decreases.

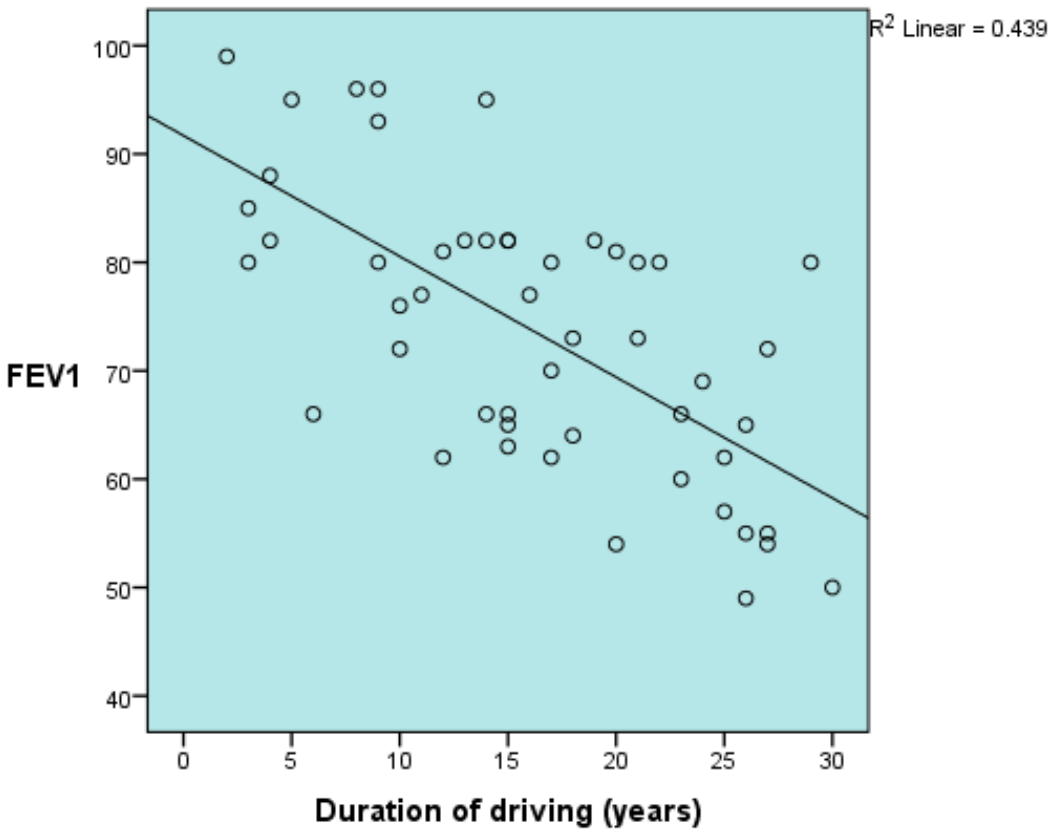

Figure no : 16 Scatter plot showing the relation between pulmonary function parameter FEV1 and duration of driving among subjects

It was observed from the scatter plot shown above that the pulmonary function parameter FEV1 of subjects was having a negative relationship with the duration of driving.

Table no : 13 Correlation between pulmonary function parameter FEV1and duration of driving

Correlation between FEV1and duration of driving			
		Duration of driving	**FEV1**
Duration of driving	Pearson Correlation	1	-0.663**
	Sig. (2-tailed)		0.000
	Total	50	50
FEV1	Pearson Correlation	-0.663**	1
	Sig. (2-tailed)	0.000	
	Total	50	50
**. Correlation is significant at the 0.01 level (2-tailed).			

Pearson correlation shows that there is statistically highly significant negative relationship between pulmonary function parameter FEV1 and duration of driving ($r = -0.66$, $p<0.01$), i.e. as the duration of driving increases, FEV1 of subjects decreases.

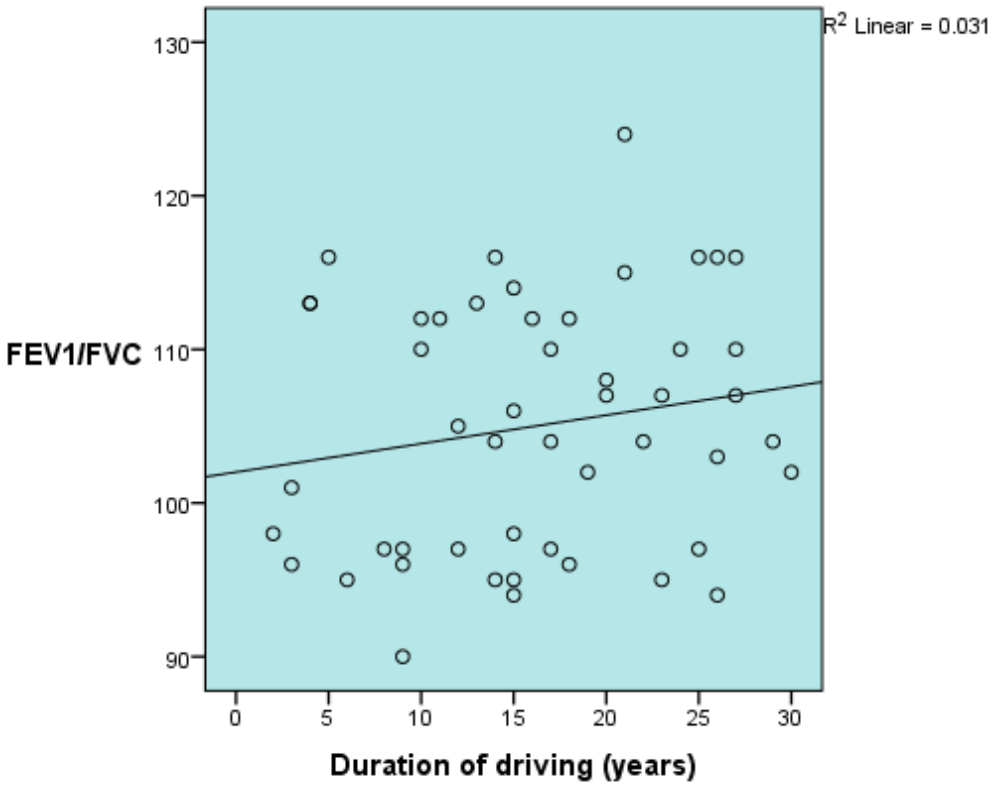

Figure no : 17 Scatter plot showing the relation between pulmonary function parameter FEV1/FVC and duration of driving among subjects

It was observed from the scatter plot shown above that the pulmonary function parameter FEV1/FVC of subjects was having a positive relationship with the duration of driving.

Table no.:14 Correlation between pulmonary function parameter FEV1/ FVC and duration of driving among subjects

Correlation between FEV1/ FVC and duration of driving			
		Duration of driving	**FEV1/FVC**
Duration of driving	Pearson Correlation	1	0.176
	Sig. (2-tailed)		0.223
	Total	50	50
FEV1/FVC	Pearson Correlation	0.176	1
	Sig. (2-tailed)	0.223	
	Total	50	50

Pearson correlation shows that there is statistically non-significant positive relationship between pulmonary function parameter FEV1/FVC of subjects and duration of driving ($r = 0.18$, $p>0.05$), i.e. as the duration of driving increases, FEV1/FVC also increases.

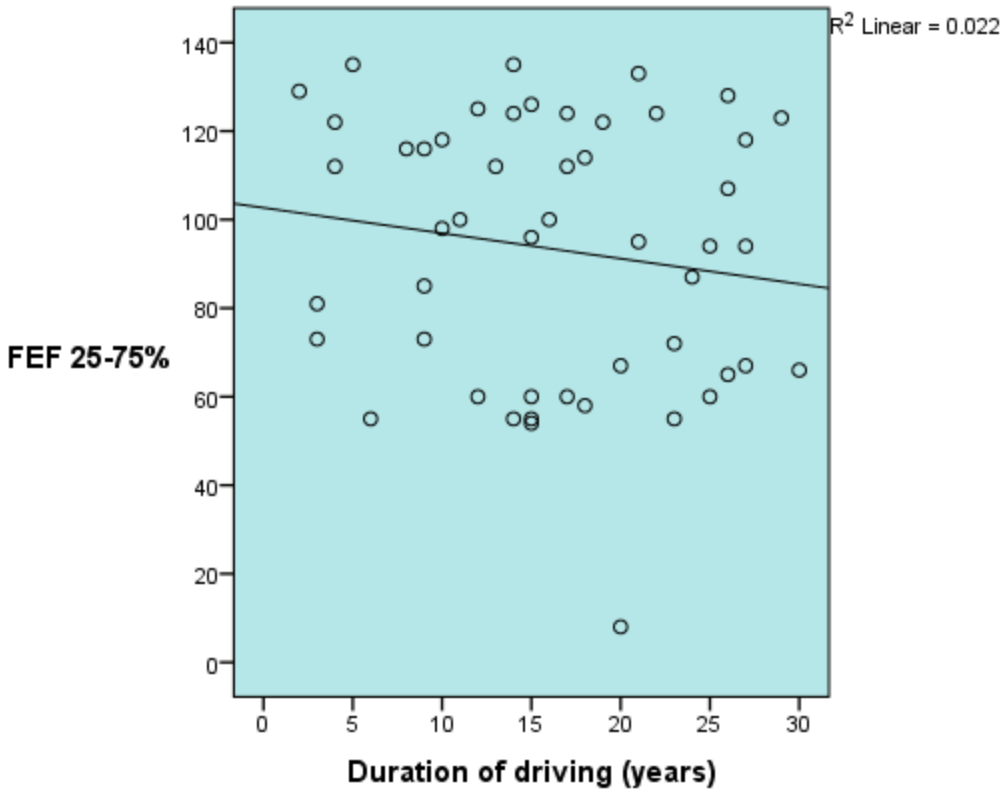

Figure no : 18 Scatter plot showing the relation between pulmonary function parameter FEF 25-75% and duration of driving among subjects

It was observed from the scatter plot shown above that the pulmonary function parameter FEF 25-75% of subjects was having a negative relationship with the duration of driving.

Table no : 15 Correlation between pulmonary function parameter FEF25-75% and duration of driving among subjects

Correlation between FEF25-75% and duration of driving			
		Duration of driving	**FEF 25-75%**
Duration of driving	Pearson Correlation	1	-0.147
	Sig. (2-tailed)		0.307
	Total	50	50
FEF25-75%	Pearson Correlation	-0.147	1
	Sig. (2-tailed)	0.307	
	Total	50	50

Pearson correlation shows that there is statistically non-significant negative relationship between pulmonary function parameter FEF 25-75% of subjects and duration of driving ($r = -0.15$, $p>0.05$), i.e. as the duration of driving increases, FEF 25-75% of subjects decreases.

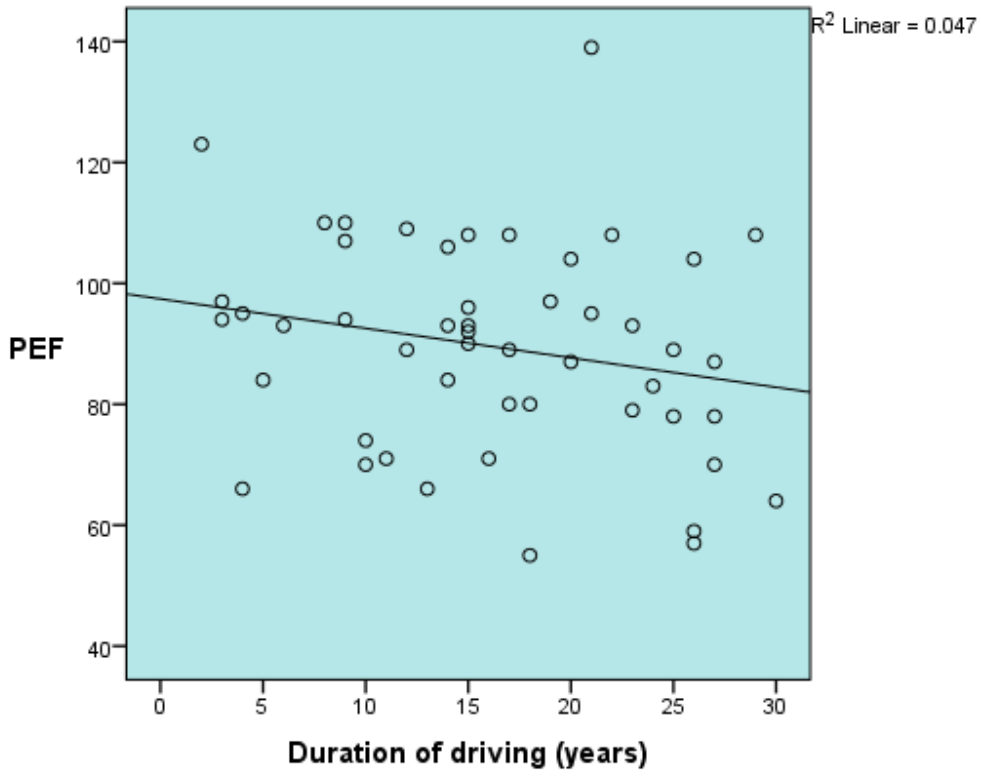

Figure no : 19 Scatter plot showing the relation between pulmonary function parameter PEF and duration of driving among subjects

It was observed from the scatter plot shown above that the pulmonary function parameter PEF of subjects was having a negative relationship with the duration of driving.

Table no : 16 Correlation between pulmonary function parameter PEF and duration of driving among subjects

Correlation between PEF and duration of driving			
		Duration of driving	**PEF**
Duration of driving	Pearson Correlation	1	-0.216
	Sig. (2-tailed)		0.132
	Total	50	50
PEF	Pearson Correlation	-0.216	1
	Sig. (2-tailed)	0.132	
	Total	50	50

Pearson correlation shows that there was statistically non-significant negative relationship between pulmonary function parameter PEF of subjects and duration of driving ($r = -0.22$, $p>0.05$), i.e. as the duration of driving increases, PEF decreases.

DISCUSSION

Nature maintains a balance between land, water, air and all living organisms in the world. Any kind of imbalance in the biosphere results in environmental pollution.

Rapid industrialization, urbanization, use to motor vehicles, agriculture, nuclear energy program are the major causes of environmental pollution in the world. Experimental studies indicate that due to airborne contaminants of diesel fumes, changes in PFTs are seen due to injury to airways and parenchyma in subjects who are exposed to it, because lungs are the major site of contact between the body and the environment.

The auto drivers were the susceptible persons as most of the time they are busy on the roads and were exposed to automobile exhaust and other air pollutants. Exposure to air pollutants over a long period is said to have deleterious effects on the respiratory functions of auto drivers.

In the present study estimation of various lung parameters like FVC ,FEV1, FEV1/FVC, FEF25-75%,PEF were done using Easy on PC spirometry among auto drivers of age 20-50 years who were non smokers and compared with the normal population.

We have compared the various lung function parameters with respect to the duration of driving of auto drivers. Based on the duration of driving they

were divided into three groups. Persons driving for 2-10 years,11-20 years and more than 20 yrs.

Figure 10 shows there was no statistically significant difference in age, height ,weight & BMI between control and study group ruling out the confounding variables regarding Biophysical profile.

FVC, FEV1:

Figure 11 shows there is decrease in mean of respiratory function parameters like FVC and FEV1 among subjects when compared to the control group. In this study FVC is reduced to less than 80% which is of restrictive type of impairment.

The frequency of bronchitis had close relation with age, amount and duration of cigarette smoking. In bidi smokers it was observed that there is higher incidence of decreased FEV1 in older subjects. There was higher percentage of subjects in cigarette smokers having abnormal FEV1[74]

The dust particles in the inhaled air which are ladened with bacteria is prevented from reaching the alveoli by ciliary escalator action, but the particles < 2 micro meter in diameter reaches the alveoli[69]. Diesel exhaust particles have size < 0.1 micrometer by releasing reactive oxygen species produces inflammation in the lungs and affects its function.

Diesel exhaust particulate constitutes a large proportion of the PM in ambient air. In particular, diesel exhaust fumes cause bronchoconstriction,

neutrophilic inflammation and dysfunction of alveolar phagocytosis together with histamine release from mast cell in healthy individuals[70].

This is similar to study conducted in pune city by **Gavali et al[59]** which shows reduction in FVC functions in auto drivers which is of restrictive type of lung impairment when compared to control group. They also concluded that those who are driving more than 5 years have very highly significant reduction in their FVC. In their study they had not compared the other lung parameters between the groups whereas in our study we compared other parameters also which confirms restrictive pattern of lung diaease.

Human lung parenchyma retain PM<2.5 (the so called respiratory PM). PM is a highly toxic material because of its small size chemical composition, as suggested by the influx of inflammatory leukocytes into the airspace (30-x). PM has a variety of effect on lung defences. Transition metals contained in PM, particularly iron, damage the airways by generating free radicals and stress[71]. The main cells involved in the initial pro-inflammatory responses to particles are the macrophages and induces oxidative stress only in these cells. Therefore chronic exposure to them can lead to inflammation of respiratory tract and lung parenchyma. These contribute to substantial decrease in lung functions.

FEV1/FVC:

In the present study, the FEV1/FVC ratio is normal and did not show any significant difference among auto drivers when compared with controls. FEVI/FVC ratio is a more sensitive indicator of airway obstruction than FVC or

FEV1 alone. This shows that the auto drivers showed restrictive type of pulmonary impairment as evidenced by significant reduction in FEV1, FVC and normal FEV1/FVC ratio. This may be due to continuous exposure of diesel exhaust pollutants which in turn affects their respiratory functions.

Table 7 & 8 shows there is statistically highly significant association between FVC, FEV1 with the duration of driving among subjects. There is gradual decrease in the values of FVC and FEV1 with drivers driving more than 10 years and 20 years when compared to the persons driving less than10 years.

This is similar to the study conducted by Chattopadhyay et al[68] who assessed the respiratory functions on garage workers, drivers and conductors in Kolkata city and found that FEV1, FEV1% FEF25%-75%and flow rates showed gradual decrease with increase in their duration of exposure. Restrictive, obstructive and combined types of impairments were noticed. But the respiratory impairments were mostly of restrictive type. They concluded the effect of pollution by dust and fumes may be responsible for these pulmonary function impairments, restrictive impairments being greater.

Generally the drivers work more than 8 hours a day. The respiratory morbidity attributed to diesel exhaust exposure is high in such individuals. There is excessive decrement in the lung function when the persons have sustained work in that particular environment which is the reliable indicator for occupational exposure.

Another study done by **Afshan afroz et al** in Gulbarga city where they compared FVC, FEV1 of non smoking auto rickshaw drivers driving for more than 5 years with non drivers. There was a highly significant decrease in FVC and FEV1 but their FEV1/FVC is above 88% which is normal showing restrictive pattern of lung impairment among study group.

Exhaust from automobile consists of complex mixture of particulate matter and different gases. These includes oxides of nitrogen, sulphur dioxide, carbon monoxide, hydrocarbons and particulate matter. Combined exposure of particulate matter along with the exposure of irritant gas such as nitrogen dioxide results in increased damage to lung when exposed either substances individually which has been demonstrated in various animal studies[69].

Along with particulate pollutants, SO2 and NO2 have a greater chance to reach the deeper parts of the lungs. The properties and concentration of surfactant are altered by these gaseous pollutants and may thus contribute to the early closure of smaller airways. Most of the terminal bronchioles may be compromised before other pulmonary function tests such as FEV1 are affected.[71]

All these reasons in combination with respiratory muscle weakening effect due to effects of carbon monoxide[73], can explain the significant decrease in the lung function test parameters, primarily restrictive pattern of lung disease as observed in our study.

FEF 25-75, PEF:

Figure 18 & 19 shows that there is negative correlation between FEF 25-75%, PEF with the duration of driving. This shows that they have restrictive pattern of lung disease rather than involvement of smaller airways.

Similar study conducted in auto rickshaw drivers of Aurangabad showed that there was significant decline in lung functions in auto rickshaw drivers as compared to control group. It was observed that FVC and FEV1 in auto drivers though less than control group but was in normal limits. FEV1% and FEF 25-75 of auto drivers was significantly less than control group which exhibit obstructive type of lung disease which is contrary to our study. Duration of auto driving had no relation with decrease in lung functions as duration considered in study was less than 10 years. This may be possibly due to difference of methodology adopted to analyse the spirometric data.

Histopathological studies have showed evidence that the smaller airways are the major site of damage in persons living in areas of high air pollution.[72] Diesel exhaust generates particles are extremely small which is of size 0.02-0.2 nanometer .These small sized particles with their greater surface area can carry much larger fraction of hydrocarbons and metals on their surface which are toxic compounds. Moreover they remain in the air for the longer period of time and they also get deposited in lungs in greater amounts than larger sized particles[72].

Similar study among three wheeler taxi drivers was done at Bikaner city[60]. They found that all the five parameters were reduced except FEV1/FVC ratio that was normal and indicated restrictive lung impairment. The reduced values of PEFR indicate obstructive lung impairment. While the values of FVC, FEV1 and FEF25–75% were reduced significantly and indicated mixed restrictive and obstructive impairment. Mixed picture of restrictive and obstructive lung impairment was prevalent amongst taxi drivers (study group). They compared the lung functions between smokers and non smokers.In their study there was a fall in percentage predicated FVC in study group as compared to control both in non-smokers and smokers

CHANGES WITH DURATION OF DRIVING:

Figure 12 &13 shows the bar diagram of the percentage predicted value with respect to duration of driving which shows the subjects who had been driving for a longer duration had lower percentage predicted values of FVC, FEV1 when compared to controls.

This reduction in values is not due to increasing age of subjects since each parameter was analysed as percentage predicted value for that particular age and BMI thus preventing age related decline in spirometric values.

Table 15 & 16 shows there is statistically highly significant negative relationship between the duration of driving and FVC, FEV1, which decreases as the duration of driving increases.

This was similar to the study conducted in non smoking auto rickshaw drivers in kerala done by **Ibrahim Farooque etal**[58]and compared with controls. All the lung function parameters were reduced significantly in the auto rickshaw drivers as compared to control subjects in the same age group and socio economic status. They also found that respiratory function tests of auto rickshaw drivers who had worked for more than 10 years were more affected than those who had worked for less than 10 years. Majority of their subjects were found to have mixed obstructive and restrictive lung impairment.

LIMITATIONS

- The limitation of the present study is the small sample size of subjects, which is not ideal for cross-sectional analysis and thus the statistical significance of the results should be interpreted with caution. The present study can be used as basis for planning longitudinal or cross sectional studies with a larger sample size.

- As we including only non-smoking auto-rickshaw drivers, we were not able to assess as the effects of air pollution on the lung function of smoking auto-rickshaw drivers, who form the majority of auto-rickshaw drivers. As smoking is the individual risk factor affecting the lung function, accelerated decline in lung parameters occurs which is to be evaluated.

- Further studies using a larger sample size with a cohort study model with analysis of the effect of preventive measures against air pollution on lung function is required.

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CONCLUSION

Pulmonary function parameters of 50 non smoking auto drivers who were driving for more than 2 years were compared with age matched 50 controls.

From the present study it was concluded

- The auto drivers were having a significantly reduced respiratory parameters like FVC, FEV1 and normal FEV1/FVC when compared with control groups whose age, weight and height are matched.
- There is significant difference in the above values concluding the pattern of lung disease being restrictive type.
- Also it was observed that auto drivers who were driving for more than 10 years have their respiratory functions affected more when compared with those who were working less than 10 years.
- Our study confirms that long term exposure to outdoor air pollution along with vehicle exhaust results in reduction of lung functions of the auto rickshaw drivers especially of restrictive type of lung impairment.

Various measures can be taken to reduce the ill effects of pollutants on auto drivers by using personnel protective equipment and the use of LPG fuel can be encouraged which is said to be low emitting fuel or solar panel autorickshaws.

They can be health educated to have face masks, physical exercise, periodic monitoring of respiratory functions and to have regular health check ups.

Quality of air standards should be checked and the levels of pollutants should be kept below the maximum permissible levels.

SUMMARY

- Exposure to vehicle exhaust affects the health of an individual, especially the respiratory system being primary target of all inhaled pollutants.
- This study was done to evaluate the involvement of lung structures among auto drivers who were exposed to vehicle exhaust.
- The study was conducted in 50 nonsmoker auto drivers who were driving for more than 2 years and 50 age matched healthy controls. The study was done in the department of physiology. After obtaining ethical committee clearance and informed consent from the participants spirometry was done using Easy On PC spirometer.
- Both Flow vs Time Graph and Flow Volume Loop were recorded. Percentage of predicted values of FVC, FEV1, FEV1/FVC AND FEF 25-75% were taken for analysis.
- The results showed that there is significant decrease in respiratory parameters resulting in restrictive pattern of lung disease among auto drivers and there was highly significant decrease in lung parameters with increasing duration of driving.
- Hence auto drivers are highly susceptible to the effects of automobile air pollution when constantly exposed, measures has to be taken to prevent such hazards.

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DATA COLLECTION FORM

Name :

Age :

Sex :

Height :

Weight :

Occupation :

Socioeconomic status :

Present complaints with duration :

Past history :

- H/o drug intake
- H/O cardiovascular disease
- H/O any respiratory illness
- H/O lung surgery
- H/O connective tissue disease
- H/O diabetes mellitus / hypertension

Personal history:

- H/O smoking
- H/O alcoholism
- H/O tobacco chewing

Occupational history:

Family history:

- H/O any respiratory illness among family members.

Clinical examination:

- Vital signs
- General examination
- Examination of respiratory system
- Examination of cardiovascular system

Investigation:

- spirometry

PATIENT CONSENT FORM

STUDY DETAIL :

"**EVALUATION OF RESPIRATORY IMPAIRMENT BY PULMONARY FUNCTION TEST IN AUTO DRIVERS"**

STUDY CENTRE:

Department of Physiology, Chengalpattu Medical College, Chengalpattu.

INDIVIDUAL NAME: **AGE:** **SEX:**

IDENTIFICATION NUMBER:

I confirm that have understood the purpose of procedure for the above study.

I have the opportunity to ask question and all my questions and doubts have been answered to my satisfaction.

I understand that my participation in the study is voluntary and that I am free to withdraw anytime without giving any reasons, without my legal rights being affected.

I understand that my investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study, I understand that my identity

will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arrives from the study.

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team.

I hereby give consent to participate in this study.

I hereby give permission to undergo complete clinical examination and diagnostic test.

Signature of investigator

Signature / Thumb impression of participant

Date:

Place:

Participant's Address

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யபடும் தலைப்பு : ஆட்டோ ஓட்டுனர்களிடம் நுரையீரல் பாதிப்பு உள்ளதா என்பதை ஸ்பைரோமெட்ரி மூலம் பரிசோதித்து மதிப்பீடு செய்தல்

ஆய்வு செய்யபடும் இடம் :

பங்கு பெறுபவரின் பெயர் :

பங்கு பெறுபவரின் வயது : பங்கு பெறுபவரின் எண்:

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விபரங்கள் எனக்கு விளக்கப்பட்டது. நான் இவ்வாய்வில் தன்னிச்சையாக பங்கேற்கிறேன் எந்த காரணத்தினாலோ எந்த சட்டச்சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகிக் கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.

இந்த ஆய்வு சம்பந்தமாகவோ இதை சார்ந்து மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொண்டேன். இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ முடிவையோ பயன்படுத்திக் கொள்ளமாட்டேன்.

இந்த ஆய்வில் பங்குகொள்ள ஒப்புக் கொள்கிறேன். இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என உறுதியளிக்கிறேன்.

பங்கேற்ப்பவரின் கையொப்பம்	**சாட்சியாளரின் கையொப்பம்**
இடம்	இடம்
தேதி	தேதி

MASTER CHART- CONTROL GROUP

S.No	Name	Age	Sex	Ht	Wt	FVC %	FEV 1	FEV1/FVC	FEF 25-75%	PEF	Pattern	Smoking
1	Ajay	22	M	164	74	97	99	102	136	146	normal	ns
2	Akilan	21	M	163	64	103	93	90	85	107	normal	ns
3	Hari	31	M	160	72	106	98	92	88	110	normal	ns
4	Manoj	21	M	179	85	81	87	109	98	97	res mild	ns
5	Sasi	43	M	157	54	66	64	96	58	55	normal	ns
6	Elavarasan	20	M	162	45	83	80	96	73	94	normal	ns
7	Senthil	40	M	170	65	70	66	95	55	93	res mild	ns
8	Vijay	30	M	174	62	102	99	98	129	123	normal	ns
9	Velu	34	M	174	67	105	102	95	120	99	normal	ns
10	Shankar	44	M	152	50	119	115	100	140	134	normal	ns
11	Moorthy	32	M	173	53	99	96	97	116	110	normal	ns
12	Sundar	37	M	169	56	92	94	103	125	104	normal	ns
13	Prabakar	42	M	178	58	77	81	105	125	109	res mild	ns
14	Santhosh	30	M	168	55	97	99	102	136	146	normal	ns
15	Jothi	34	M	172	78	51	54	107	67	87	res mild	ns
16	Ganesh	45	M	170	54	100	95	96	115	109	normal	ns
17	Vinoth	22	M	172	55	96	98	101	134	145	normal	ns
18	Balaji	26	M	172	55	98	94	95	115	108	normal	ns
19	Varun	23	M	165	64	102	92	90	84	105	normal	ns
20	Ramesh	43	M	178	67	76	80	105	125	108	res mild	ns
21	Raja	42	M	164	75	74	81	108	98	103	res mild	ns
22	Gopi	28	M	162	65	102	92	90	85	107	normal	ns
23	Ravi	32	M	172	72	60	63	104	72	63	res mild	ns
24	Manikam	38	M	174	64	103	98	96	127	120	normal	ns
25	Kumar	28	M	175	50	83	80	94	74	96	normal	ns

S.No	Name	Age	Sex	Ht	Wt	FVC %	FEV 1	FEV1/FVC	FEF 25-75%	PEF	Pattern	Smoking
26	Velayutham	40	M	172	60	98	97	96	115	108	normal	ns
27	Velmurugan	39	M	169	65	91	95	91	87	109	normal	ns
28	Vivek	36	M	168	58	97	99	102	135	143	normal	ns
29	Mahendran	43	M	176	57	76	80	104	126	108	res mild	ns
30	Satish	29	M	173	62	101	97	96	127	120	normal	ns
31	Naveen	20	M	184	98	103	90	87	89	104	normal	ns
32	Shriram	22	M	182	74	112	100	89	104	120	normal	ns
33	Ganapathy	32	M	174	70	109	98	89	90	120	normal	ns
34	Gopinath	29	M	172	68	117	107	92	112	137	normal	ns
35	Subravel	40	M	174	67	118	114	97	132	99	normal	ns
36	Hameed	31	M	175	76	64	62	97	60	89	res mild	ns
37	Murugappan	35	M	165	74	82	80	99	75	85	normal	ns
38	Rajavel	38	M	151	70	88	81	92	57	118	normal	ns
39	Robin	25	M	174	73	110	97	88	92	121	normal	ns
40	Natraj	33	M	180	72	110	98	89	103	119	normal	ns
41	Rangarajan	27	M	174	63	101	99	97	128	122	normal	ns
42	Sivaguru	41	M	162	44	83	81	97	73	95	normal	ns
43	Suresh	36	M	164	73	81	81	99	75	86	normal	ns
44	Chandran	37	M	174	75	117	112	96	131	99	normal	ns
45	Ajmal	34	M	182	75	112	100	89	104	120	normal	ns
46	Kalai	31	M	180	98	103	90	87	89	104	normal	ns
47	Rajgopi	23	M	174	70	117	107	92	112	137	normal	ns
48	Sivakumar	29	M	179	88	103	90	87	89	104	normal	ns
49	Shanmugam	39	M	160	59	119	117	100	140	134	normal	ns
50	Saravanan	27	M	169	74	82	80	99	75	85	normal	ns

MASTER CHART – STUDY GROUP

S.no	Name	Age	Occu.(yrs)	Ht in cm	Wt in kg	FVC	FEV1	FEV1/FVC	FEF 25-75%	PEF	Pattern
1	manimaran	28	9	169	85	83	80	96	73	94	normal
2	nagalingam	39	14	176	69	70	66	95	55	93	mild restriction
3	gopalakrishnan	50	25	170	70	64	62	97	60	89	mild restriction
4	kamalakannan	50	30	167	65	49	50	102	66	64	mod restriction
5	periyasamy	46	17	173	74	64	62	97	60	89	mild restriction
6	baskaran	49	29	180	85	77	80	104	123	108	mild restriction
7	gunasekaran	49	23	174	74	69	66	95	55	93	mild restriction
8	mathivanan	48	26	166	77	48	49	103	65	57	mod restriction
9	shanmugam	26	6	173	97	70	66	95	55	93	mild restriction
10	a.george	29	9	170	65	103	93	90	85	107	normal
11	sekar b	44	25	166	74	50	57	116	94	78	mod restriction
12	ravikumar	49	21	170	63	73	80	115	95	95	mild restriction
13	jaikumar	39	19	170	86	76	82	102	122	97	mild restriction
14	kannan	28	5	174	80	89	95	116	135	84	normal
15	gopinath	49	23	167	70	61	60	107	72	79	mild restriction
16	velu	26	4	166	65	81	88	113	122	95	normal
17	murugan	46	27	175	84	64	72	110	118	70	mild restriction
18	hari	43	21	156	70	64	73	124	133	139	mild restriction
19	rajesh kumar	32	12	174	84	64	62	97	60	89	mild restriction
20	jagdeesan	32	10	160	82	64	72	110	118	70	mild restriction
21	kumar	38	15	175	55	70	66	95	55	93	mild restriction
22	velmurugan	35	15	163	68	74	82	114	96	96	mild restriction
23	omkumar	39	12	175	88	77	81	105	125	109	mild restriction
24	sathik batsha	45	15	158	84	69	65	94	54	92	mild restriction
25	shanmugam	50	27	158	65	51	54	107	67	87	mod restriction

S.no	Name	Age	Occu.(yrs)	Ht in cm	Wt in kg	FVC	FEV1	FEV1/FVC	FEF 25-75%	PEF	Pattern
26	babu	38	18	157	58	65	73	112	114	80	mild restriction
27	vasu	27	3	165	63	85	85	101	81	97	normal
28	kumaravel	37	13	167	82	73	82	113	112	66	mild restriction
29	govindaraj	23	3	168	77	83	80	96	73	94	normal
30	sabapathy	45	20	170	90	75	81	108	8	104	mild restriction
31	mohan	36	15	165	55	65	63	98	60	90	mild restriction
32	dhanasekaran	41	22	165	63	76	80	104	124	108	mild restriction
33	kumar	37	17	162	70	64	70	110	112	80	mild restriction
34	raman	45	20	180	100	51	54	107	67	87	mod restriction
35	vishwanathan	48	18	160	55	66	64	96	58	55	mild restriction
36	ravikumar	50	27	176	78	48	55	116	94	78	mod restriction
37	subramani	34	11	165	63	74	77	112	100	71	mild restriction
38	moorthy	28	4	165	65	73	82	113	112	66	mild restriction
39	venkatasen	35	16	170	72	74	77	112	100	71	mild restriction
40	somasundaram	30	8	160	65	99	96	97	116	110	normal
41	ruban	25	2	173	78	102	99	98	129	123	normal
42	krishnamoorthy	50	24	174	68	63	69	110	87	83	mild restriction
43	jothilingam	36	14	174	65	89	95	116	135	84	normal
44	palani	43	10	176	100	74	76	112	98	74	mild restriction
45	thigarajan	50	26	182	95	69	65	94	128	104	mild restriction
46	vetrivel	37	15	162	58	78	82	106	126	108	mild restriction
47	rajasekaran	38	17	175	58	77	80	104	124	108	mild restriction
48	vasanth	50	26	169	85	47	55	116	107	59	mod restriction
49	vignesh	36	14	166	70	77	82	104	124	106	mild restriction
50	arul	31	9	165	73	99	96	97	116	110	normal